ANNUAL SESSION • CHICAGO • JUNE 9-13, 1952

This Issue Exceeds 150,000 Copies

THE JOURNAL

OF THE

American Medical Association

Volume 148, No. 16 — Published Weekly at 535 North Dearborn Street, Chicago 10, Illinois. Subscription, $15. Single Copy, 45 Cents — April 19, 1952

ORIGINAL ARTICLES

"ARMCHAIR" TREATMENT OF ACUTE CORONARY THROMBOSIS 1365
Samuel A. Levine, M.D., and Bernard Lown, M.D., Boston

ANOXIA IN MYOCARDIAL INFARCTION AND INDICATIONS FOR OXYGEN THERAPY 1370
Craig W. Borden, M.D.; Richard V. Ebert, M.D., and Russell H. Wilson, M.D., Minneapolis

HISTORY OF THE TREATMENT OF CORONARY HEART DISEASE 1372
N. C. Gilbert, M.D., Chicago

DETECTION AND TREATMENT OF NUTRITIVE FAILURE IN CHILDREN 1376
Tom D. Spies, M.D.; Samuel Dreizen, D.D.S.; George S. Parker, D.D.S., and Donald J. Silberman, M.D., Birmingham, Ala.

LIMITATIONS OF SYMPATHECTOMY IN TREATMENT OF DIASTOLIC HYPERTENSION 1382
Geza de Takats, M.D., Chicago

NATURE AND RECOGNITION OF CEREBRAL PALSY IN INFANCY 1389
Meyer A. Perlstein, M.D., and Harry E. Barnett, M.D., Chicago

FACTORS RELATING TO THE DISTRIBUTION OF PHYSICIANS 1397
H. G. Weiskotten, M.D., Skaneateles, N. Y.

ESTATE PLANNING FOR PHYSICIANS. PART 1: THE BUSINESS SIDE OF THE PHYSICIAN'S LIFE 1401
W. S. McClanahan, Chicago

REHABILITATION OF PATIENTS WITH DEEP BURNS 1405
James Barrett Brown, M.D., St. Louis

REHABILITATION OF HAND FUNCTION IN RHEUMATOID ARTHRITIS 1408
Donald L. Rose, M.D., Kansas City, Kan., and H. Worley Kendell, M.D., Chicago

CLINICAL NOTES

EARLY PERFORATION OF INTERVENTRICULAR SEPTUM AFTER MYOCARDIAL INFARCTION 1413
George D. Geckeler, M.D.; Daniel J. Marino, M.D., and John E. Gregory, M.D., Philadelphia

PNEUMATOSIS OF THE COLON 1416
Richard H. Marshak, M.D.; Joan J. Lipsay, M.D., New York, and A. I. Friedman, M.D., Hackensack, N. J.

TALC PNEUMOCONIOSIS 1418
Paul S. Friedman, M.D.; Milton A. Bell, M.D., and Leon Solis-Cohen, M.D., Philadelphia

BRONCHOGRAPHY IN IODINE SENSITIVITY 1419
Peter A. Theodos, M.D., Philadelphia

SPECIAL REPORTS

Council on Foods and Nutrition

ACCEPTED PRODUCTS 1421

EDITORIALS

Folic Acid Deficiency in Macrocytic Anemia of Pregnancy and Infancy 1422
Surgery in Patients with Cortisone-Induced Adrenal Insufficiency 1422
Procaine in Athletic Injuries 1423

ORGANIZATION SECTION

Report of Board of Trustees 1424
The Federal Government as the Insured of a Private Insurer 1426
Federal Medical Legislation 1427
State Medical Legislation 1428

REGULAR DEPARTMENTS

Medical News 1429
Meetings 1433
Examinations and Licensure 1434
Deaths 1435
Government Services:
Public Health Service 1438
Miscellaneous 1438
Foreign Letters:
Belgium 1439
Italy 1440
London 1440
Spain 1441
Sweden 1442
Correspondence:
Anesthesia in Operation for Pheochromocytoma. Coarctation of the Aorta. Warfarin for Suicide. 1443
Medicolegal Abstracts 1444
Medical Literature Abstracts 1445
Book Reviews 1454
Queries and Minor Notes:
Influenza. Treatment of Calcinosis of Kidneys. Iron and Black Stools. Cold Disinfection of Instruments. Transmission of Deaf Mutism. Removal of Acne Scars. Prostatic Cancer. Irradiation from Television Receivers. Hypertrophy of Both Breasts. Nasopharyngeal Fibroma. Undescended Testis. Eosinophilic Granuloma of Bone. Substernal Thyroid Adenoma. Morphine Injection Using Unsterile Tablet. The Ophthalmoscope. Cracks at Corner of Mouth. Sphenopalatine Ganglion Neuralgia. ..1458

WASHINGTON NEWS

Proposed Federal Department of Health. President Truman's Health Commission. The Korean Situation. Deferment of Medical Students Recommended. Certification of Causes of Death. Adv. Page 13
National Medical Societies ..... Adv. Page 18
Tonics and Sedatives ..... Adv. Page 30
Medical Education Foundation ..... Adv. Page 41
Books Received ..... Adv. Page 45

Cecil & Loeb's Textbook of Medicine

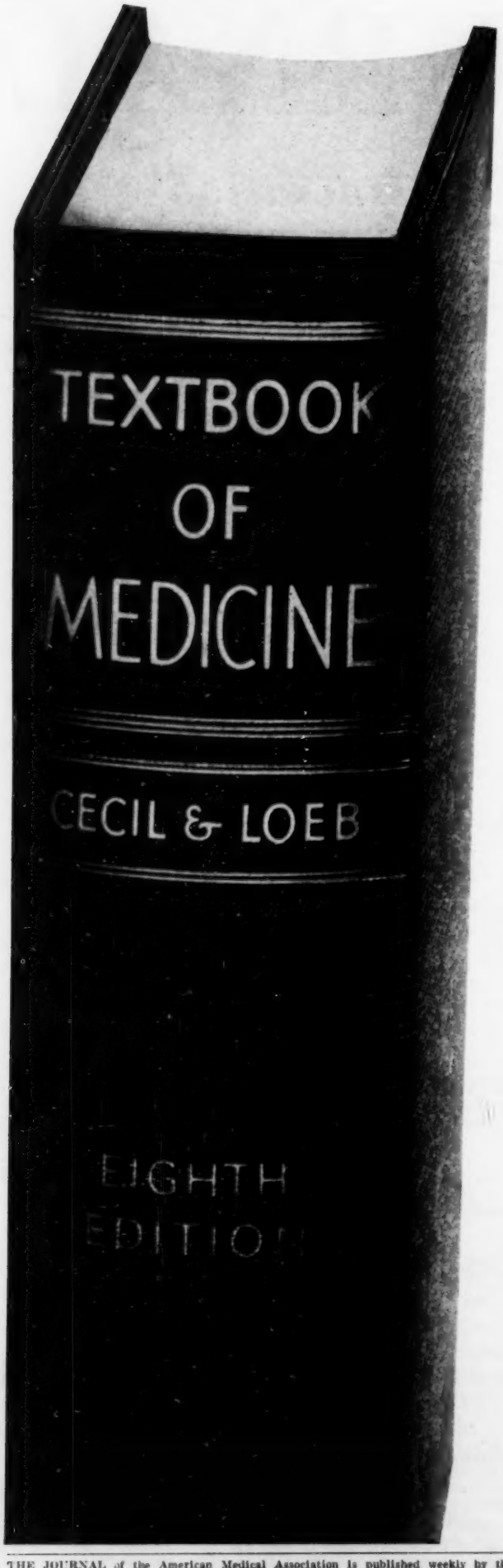
The *New (8th) Edition* of this standard textbook covers 809 separate diseases and disorders, with thorough discussions of etiology, pathologic physiology, symptomatology, diagnosis (including laboratory tests), prognosis, and treatment. Psychologic aspects are carefully considered.

Few textbooks are esteemed as highly by the profession as this one. In the 25 years of its existence, it has been used by almost 300,000 students and practicing physicians. This *New (8th) Edition,* with its 168 outstanding contributors, is the most complete and useful that has ever appeared.

For this *New (8th) Edition,* DR. ROBERT F. LOEB, of Columbia University, has shared with DR. RUSSELL L. CECIL the responsibility of editorial leadership.

See SAUNDERS Advertisement on next two pages

H M POLLARD MD
2012 VINEWOOD BLVD
ANN ARBOR MICH



"Big" books in

one friend who will tell you

CECIL'S MEDICINE has been friend and advisor to almost 300,000 physicians during the past 25 years. *And it was a rare occasion when this great book failed to supply the answer requested of it.*

The *New (8th) Edition* upholds the fine tradition. Remarkably complete (809 separate diseases are covered); unquestionably authoritative (contributions by 168 recognized authorities); and thoroughly up-to-date—it will serve you even more fully than has its predecessors.

By 168 American Authorities. Edited by RUSSELL L. CECIL, M.D., Cornell University; and ROBERT F. LOEB, M.D., Columbia University. 1627 pages, 7" x 10", with 204 illustrations. $13.00.

New (8th) Edition

Use

SAUNDERS

Order Form

THE JOURNAL of the American Medical Association is published weekly by the American Medical Association. Subscription price, $15.00 a year, 45c a copy. Canadian $16.50. Foreign $19.00. Entered as second class matter June 25, 1885, at the Postoffice at Chicago, Ill., under act of March 3, 1879. Address all communications to American Medical Association, 535 N. Dearborn St., Chicago 10, Illinois

THE JOURNAL
of the American Medical Association
Published Under the Auspices of the Board of Trustees

VOL. 148, NO. 16 CHICAGO, ILLINOIS APRIL 19, 1952
COPYRIGHT, 1952, BY AMERICAN MEDICAL ASSOCIATION

"ARMCHAIR" TREATMENT OF ACUTE CORONARY THROMBOSIS

Samuel A. Levine, M.D.
and
Bernard Lown, M.D., Boston

Rest for a diseased organ, be it a fractured limb or a tuberculous lung, is a therapeutic principle validated by clinical experience. It is believed that activity, with its complex biochemical and physical demands, diverts from the reparative process the available energy essential for optimal healing. When the heart is diseased, the only feasible application of the principle of rest is the attempted diminution of the cardiac load. Rest in bed has been traditionally regarded as tantamount to optimal rest for the heart. This is the practice in congestive heart failure and finds its most rigid application in acute coronary thrombosis, when the patient is placed flat in bed for three to six weeks.[1]

It has been our view that recumbency in bed affords less rest to the heart than the sedentary position in a chair with the feet down.[2] The recumbent position permits maximal venous return from the periphery with associated increases in blood volume, venous pressure and ventricular diastolic filling, and augmented volume work for the heart. While the sitting position permits gravity to mobilize fluid into the dependent parts of the body, the recumbent position encourages the pooling of fluid in the pulmonary circuit. The former is essentially harmless; the latter may be disastrous. The abruptness of the onset of coronary thrombosis, with its grave prognostic connotations, afflicting as it frequently does the highly active and previously healthy person, when coupled with long-continued bedrest, saps morale, provokes desperation, unleashes anxiety, and ushers in hopelessness with respect to resumption of normal living. The deleterious physical equivalents of such emotional disquiet and psychological tension, though not measurable, are evident to both the physician and the family. A host of other harmful sequelae of bedrest have been emphasized, such as inanition, constipation, thrombophlebitis, osteoporosis, negative nitrogen balance, hypostatic pneumonia, atelectasis, and prostatic difficulties.[3] In short, the bed is not a resting place for the patient with cardiac disease.

In recent years, one of us has observed patients with acute coronary thrombosis who were becoming worse despite all known measures and then were benefited immediately and remarkably after being placed in a chair. The above-cited theoretical objections, combined with such gratifying experience, have induced us, during the past year and a half, to modify the treatment of acute coronary thrombosis. We believe that by getting these patients promptly out of bed and into a chair, we achieve more rest for the heart than is attained with traditional procedures. This is a preliminary report of results of the "armchair" treatment in 81 patients.

MATERIAL AND METHODS

Of the 81 patients with acute coronary thrombosis, 57 were admitted to the medical service of the Peter Bent Brigham Hospital and 24 were treated at home or in other hospitals. In all instances, diagnosis was established on clinical as well as electrocardiographic data. In the majority, 12 lead serial tracings showed characteristic changes of the acute process. Excluded from evaluation are patients with mild symptoms in whom the findings were explicable on some other basis or those in whom doubt was entertained as to the correctness of the diagnosis. Chart 1 summarizes some of the clinical features in the 81 cases at the time of admission. There is no doubt that this was a selected group. Among other evidence for this is the high frequency of paroxysmal arrhythmias. That the condition was not mild in this group is attested by the fact that nearly half the patients had

This study was aided in part by a United States Public Health grant (H 445).
Read before the Association of American Physicians, Atlantic City, N. J., May 1, 1951.
From the Medical Clinic of the Peter Bent Brigham Hospital and the Department of Medicine, Harvard Medical School.

1. Levine, S. A.: Coronary Thrombosis: Its Various Clinical Features, Medical Monographs, Baltimore, Williams & Wilkins Company, 1929, vol. 16. White, P. D.: Heart Disease, Macmillan Medical Monographs, ed. 3, New York, The Macmillan Company, 1944. Levy, R. L.: Diseases of Coronary Arteries and Cardiac Pain, New York, The Macmillan Company, 1936. Friedberg, C. K.: Diseases of Heart, Philadelphia, W. B. Saunders Company, 1949.
2. Levine, S. A.: Some Harmful Effects of Recumbency in Treatment of Heart Disease, J. A. M. A. **126**: 80 (Sept. 9) 1944.
3. Dock, W.: Evil Sequelae of Complete Bed Rest, J. A. M. A. **125**: 1083 (Aug. 19) 1944.

pulmonary edema and a fifth of all the patients were rapidly digitalized after entering the hospital. Whether this is a proper method of treatment is open to question.

The majority of patients were out of bed during the first two days. They were helped out of bed and placed into a comfortable mobile chair, with care taken so that no pressure was exerted on the popliteal spaces. They remained in a chair until they experienced fatigue. Our goal was to have these patients out of bed as much of the day as was possible without discomfort to them. This was achieved in some who were in the chair most of the day from the beginning of their illness. For the majority, however, this usually meant that they were out of bed about one to two hours during the first day with increasing time intervals thereafter, so that by the end of the first week they spent the larger portion of the day in a chair. The contraindications to use of the chair were (1) a continuing state of shock, (2) marked debility, and (3) concomitant cerebrovascular accident. High temperature, severe pain, a friction rub, a diastolic gallop, heart block, cardiac arrhythmias, or the need for oxygen were not regarded as contraindications to such treatment. Nearly all patients fed themselves and were either permitted the use of the bedside commode or granted toilet privileges. Anticoagulants were administered to all but nine patients. Aside from the elevation of the headposts of the bed by 8 to 9 in. blocks,[4] routine care for coronary disease was given. A few steps were allowed by the end of the third week, and hospitalization was maintained for about four weeks.

SEX	Male Female	68 13	84% 16%
AGE, YEARS	Range	42—80	
	Mean	58.4	MALE—57.3 FEMALE—64.0 LIVING—56.0 DEAD—73.0
PREVIOUS CORONARY ARTERY DISEASE	Angina Pectoris	45	54.8%
	Coronary Thrombosis	26	32.1%
COMPLICATIONS ON ADMISSION	Pulmonary edema	39	moderate—30 severe — 9
	Shock	10	
	Arrhythmias	15	
		A. fibrillation	8
		A. flutter	5
		Nodal tachycardia	4
		P.A.T.	1
		Ventricular tachycardia	2
DIGITALIZED ON ADMISSION		17	

Chart 1.—General features in 81 cases of acute coronary thrombosis. A = auricular. P.A.T. = paroxysmal auricular tachycardia.

RESULTS

Among the 81 patients, there were eight deaths, a mortality of 9.9%. Chart 2 gives data on the 73 surviving patients. Except for two patients, one of whom was out of bed on the eighth and the other on the ninth day, all the patients were in a chair within the first week in the hospital, 86% within the first three days. Convalescence was uneventful in 36 patients; i. e., these patients suffered no complications and had uninterrupted recoveries. Results were good in 27. This group, in addition to their occlusions, had either paroxysmal rapid heart action, shock, or pulmonary edema but nonetheless had a smooth course. The "remarkable" group was comprised of 10 patients who were characterized by the house staff as "critically" or "terminally" ill. They had one or more of the above-enumerated complications, and the inception of recovery appeared to coincide directly with their being placed in a chair. In the following report are described four of the 10 with striking improvement.

REPORT OF CASES

CASE 1.—F. B., a 60-year-old man, was admitted with a fresh posterolateral infarction, the second in eight months. He was acutely ill and in semicoma. His temperature was 101 F, the white blood cell count was 22,000 and a corrected sedimentation rate was 88 mm. per hour. He had a bigeminal pulse and was in shock, with a blood pressure of 35/0. His pressure was maintained with the use of phenylephrine hydrochloride. He continued to be critically ill until 40 hours later, when he was placed in a chair. He began to walk on the 21st day and was discharged on the 29th day. Having had a myocardial infarction some months before, when he was kept in bed for eight weeks, he volunteered, "What a happier time I had with this attack!" This notwithstanding the severer onset of the second episode. One year later he was in good condition. X-ray and an electrocardiogram, showed that the heart was essentially normal.

CASE 2.—S. Q., a 51-year-old man, had an anteroseptal infarction and a ventricular aneurysm due to an old coronary thrombosis. He had had two previous cerebrovascular accidents and had residual hemiplegia. He was in congestive heart failure and was regarded variously as "gravely," "critically," and "terminally" ill. Serial tracings showed auriculoventricular dissociation, then paroxysmal auricular tachycardia with block, and later auricular flutter. He was digitalized by the intravenous route.

DAY OUT IN CHAIR	1st	15
	2nd	30
	3rd	18
	4-9th	10
PROGRESS	uneventful	36
	good	27
	remarkable	10
COMPLICATIONS	6	1 HEPATITIS 2 HEMATURIA 1 FEVER 15th day 1 PAROX. TACH & C.F. 13th day 1 RECURRENT PAIN 21st day
PERSISTENCE OF ST SEGMENT ELEVATION	5	1 Aneurysm demonstration by roentgenogram 1 Roentgenogram normal 3 No roentgenogram
DURATION OF HOSPITALIZATION in 46 CASES	RANGE	15—35 days
	AVERAGE	24 days

Chart 2.—Results of "armchair" treatment of 73 patients who survived. Parox. Tach. = paroxysmal tachycardia. C.F. = congestive failure.

Despite the administration of nikethamide and oxygen, he continued to be extremely drowsy. When he was taken out of bed, on the third day, the drowsiness abated promptly; progress was then continuous. He was permitted to walk on the 25th day and was discharged on the 33rd day.

CASE 3.—E. K., a 60-year-old hypertensive woman, had a posterior myocardial infarction and a history of a previous coronary

4. Levine, S. A.: Clinical Heart Disease, ed. 3, Philadelphia, W. B. Saunders Company, 1945.

occlusion. For a day prior to admission she had an Adams-Stokes syndrome. On the day of entry she was unconscious and in shock. Her heart rate was 220, and she was proved to have a paroxysm of ventricular tachycardia. Intravenous administration of 1.3 gm. of procaine amide hydrochloride was required to stop this attack. Subsequently, auriculoventricular dissociation developed. On the third day, because of the appearance of pulmonary edema, she was placed in a chair. Improvement was rapid and uneventful.

CASE 4.—J. B., a 46-year-old man, had an extensive anterior wall infarction. For two days he continued to have excruciating chest pain. His temperature was 102 F, and he was drowsy, had profuse sweating, a friction rub, and pulmonary edema. On the second day auricular fibrillation developed. He was digitalized rapidly. He got along very poorly until he was taken out of bed, on the second day. Improvement was steady; he walked on the 19th day and was discharged on the 28th day.

COMMENT

The few complications that occurred in our series were not attributable to the "chair" treatment. There were no instances of thrombophlebitis or pulmonary embolism. The patient who had a fever episode on the 15th day had a normal chest roentgenogram and responded promptly to penicillin therapy. That pulmonary edema rarely develops after the institution of the "chair" treatment strongly suggests that this complication was actually prevented by this method. While a number of patients had angina pectoris, only one had severe pain simulating a recurrence of thrombosis. However, the electrocardiogram and laboratory studies failed to corroborate this. The most encouraging aspect of this type of care for patients with an acute coronary attack was the continued sense of well-being and high morale. This was especially apparent in those experiencing their second or third occlusion. Their invariable comment indicated that the current episode was easier to bear. When they were permitted to walk, there was no collapse, dizziness, or weakness. Convalescence proceeded without mishap, and the patient seemed prepared for every increased stress placed on the heart. Chart 3 and the table summarize the salient facts concerning the eight patients who died. Their mean age was 73.0 years, as contrasted with 56 years for the living. Three of the eight who died were women, though women constituted only 16% of the total group. This high mortality is probably due to both the older age group of the women in this study and to the greater incidence of paroxysmal rapid heart action among them. Five of the 13 women had bouts of rapid heart action. Seven of the 15 patients with paroxysmal arrhythmia died. Similar testimony as to the gravity of paroxysmal rapid heart action associated with acute coronary thrombosis has been provided in previous publications.[5] Two of the eight deaths that occurred in the entire series were attributed to pulmonary emboli, substantiated in one case at autopsy. In neither case had adequate anticoagulants been administered. Among the nine cases in this series in which anticoagulants had not been given, there were two deaths. Of the 72 cases in which either bishydroxycoumarin or heparin had been given, there were six deaths. Thus, in the group of "chair"-treated patients who also received anticoagulants, there was a mortality of 8.3%.

A certain mortality is to be expected in myocardial infarction irrespective of the type of treatment. The immediate mortality is known to be enhanced by advanced age, paroxysmal arrhythmias, heart block, shock, and congestive heart failure.[6] The eight patients who died had various combinations of the above factors. There is no evidence that the "chair" treatment was responsible for the fatalities in this group of patients.

This type of treatment of acute coronary thrombosis was first employed 14 years ago, and a report of this was made three years later.[7] The patient described was dying of severe congestive heart failure and pulmonary edema. All therapeutic measures were unavailing. He was placed in a chair in the hope that gravity would divert fluid from the lungs into the lower extremities. This was achieved, and a prompt recovery resulted. The patient was still alive some years later. During the decade that has ensued since that initial experience, physiological and clinical observations have made possible a reformulation of the principle of rest for the injured heart. Perera and

SEX	MALE	5—71.3% of ♂
	FEMALE	3—23% " ♀
AVERAGE AGE		73
TYPES OF OCCLUSIONS	POSTERIOR	3
	ANTERIOR	1
	A.S.I.	1
	A.L.I.	1
	"ROESLER DRESSLER "	2
PREVIOUS OCCLUSIONS		4
COMPLICATION on ADMISSION	PULMONARY EDEMA	7
	SHOCK	3
	PAROXYSMAL RAPID HEART ACTION	7
DIGITALIZED	ON ADMISSION	6
	MAINTENANCE	2
TIME of DEATH	1st WEEK	4
	2nd WEEK	4

Chart 3.—Analysis of the eight cases in which death occurred. A.S.I. = anteroseptal infarction. A.L.I. = anterolateral infarction.

5. Askey, J. M., and Neurath, O.: Treatment of Auricular Fibrillation Occurring with Myocardial Infarction, Am. Heart J. **30**: 253, 1945. Askey, J. M.: Auricular Flutter in Association with Myocardial Infarction, Am. J. Med. **6**: 453, 1949.

6. Rosenbaum, F. F., and Levine, S. A.: Prognostic Value of Various Clinical and Electrocardiographic Features of Acute Myocardial Infarction: Immediate Prognosis, Arch. Int. Med. **68**: 913 (Nov.) 1941.

7. Levine, S. A.: Management of Patients with Heart Failure, J. A. M. A. **115**: 1715 (Nov. 16) 1940.

Berliner [8] found that, when patients with hypertensive cardiovascular disease are placed in bed, within 12 to 24 hours there is hemodilution, a rise in blood volume, an increase in venous pressure, and a decrease in the vital capacity of the lungs. McMichael and McGibbon [9] noted that even patients without cardiac disease show a 200 cc. diminution in the vital capacity on lying down. The fact that patients with congestive heart failure do well after being placed in bed, we believe, is merely evidence of the efficacy of other therapeutic tools, such as the use of a salt-poor regimen, diuretics, digitalis, morphine, and oxygen, in counteracting the stress placed on the heart by the recumbent position.

"chair"-treated patients with coronary disease, no data are as yet available. There is no reason, however, for presuming a worsening of prognosis.

The enhanced sense of well-being was one of the most spectacular features in this series. Our inability to present this graphically does not, we believe, detract from its validity. When a patient is placed at complete bedrest after an acute coronary thrombosis, profound psychological changes follow. The patient who was active and well a few hours previously now feels as though death was hovering close by. This impression is reinforced by the physician's insistence on complete bedrest and the prohibition of even minimal movements. The

Clinical and Pathological Features of the Eight Patients (9.9% of Total) Who Died

Patient	Age, Yr.	Sex	Type of Occlusion	Previous Myocardial Infarction	Complications on Admission: Pulmonary Edema	Shock	Arrhythmia	Miscellaneous	Therapy: Anticoagulant	Digitalization	Day Out of Bed	Day of Death: Day of Hospitalization	Day After Coronary	Postmortem Findings
J. F.	74	M	"Roesler Dressler"	Yes	1+	0	Auricular fibrillation	2° heart block Uncontrolled vomiting	Yes	Yes	3rd	5	26	0
D. M.	60	M	Antero-septal	No	2+	0	Auricular flutter	Right bundle branch block	Delayed	Yes	2nd	11	16	0
J. S.	69	M	Posterior	Yes	2+	0	Auricular fibrillation Auricular flutter	2° heart block Left bundle branch block	Yes	Yes	2nd	7	10	0
M. C.	80	F	Posterior	No	1+	Yes	Auricular fibrillation Auricular flutter	Cerebrovascular accident on 3rd day	0	Yes	2nd	10	?	Acute posterior myocardial infaret Massive pulmonary embolism Cerebrovascular thrombosis
E. R.	77	F	Postero-lateral	No	3+	0	Paroxysmal auricular tachycardia	0	Yes	Yes	2nd	7	?	Acute posterior infarction No pulmonary edema
N. C.	70	F	Antero-lateral	Yes	3+	Yes	Auricular fibrillation	Old myocardial aneurysm Comatose 36 hr.	Yes	Yes	4th	18	?	Fresh myocardial infarction with cardiac rupture Old infarction and left ventricular aneurysm
L. B.	71	M	Posterior	No	1+	0	0	Marked intraventricular block	Yes	Maintenance	2nd	4	5	Occlusion of right and left circumflex arteries Post myocardial and septal infarction
M. C.	79	M	Anterior	Yes	3+	0	Paroxysmal ventricular tachycardia	0	Yes	Maintenance	2nd	4	5	0

In the evaluation of any therapeutic innovation for acute coronary thrombosis, attention must be directed to the immediate mortality and the incidence of complications. Stock must also be taken of the patient's senses of well-being during treatment and the ease of recuperation after treatment. An assessment of the usefulness of a new method must rest also on its effects on the ultimate prognosis and the patient's capacity for resuming a productive life. As regards the eventual course of the

patient thus abruptly feels he is at the mercies of forces over which he has no mastery. What is even more provocative of anxiety is the fact that he is left without any gage to assess the presence, extent, and speed of progress. That anxiety may be especially harmful to the patient with acute coronary disease may be deduced from Stead's [10] work; he found that emotion causes a marked increase in cardiac output. In contrast to this, the patient in a chair need never view himself as hopelessly ill. What is most important, however, is that the gradually increasing time out of bed provides him with the clearest index of improvement. He is made to feel he is an aware and active participant in the healing process. This awareness is more potent in allaying fear and dissipating anxiety than any of the physician's words of encouragement.

Dietrick and co-workers,[11] in their studies of the effects of long-continued recumbency on healthy human

8. Perera, G. A., and Berliner, R. W.: Relation of Postural Hemodilution to Paroxysmal Dyspnea, J. Clin. Invest. **22**: 25, 1943.

9. McMichael, J., and McGibbon, J. P.: Postural Changes in Lung Volume, Clin. Sc. **4**: 175, 1939.

10. Stead, E. A., Jr.; Warren, J. V.; Merrill, A. J., and Brannon, E. S.: Cardiac Output in Male Subjects as Measured by Technique of Right Atrial Catheterization: Normal Values with Observations on Effect of Anxiety and Tilting, J. Clin. Invest. **24**: 326, 1945.

11. Dietrick, J. E.; Whedon, G. D., and Shorr, E.: Effects of Immobilization upon Various Metabolic and Physiologic Functions of Normal Men, Am. J. Med. **4**: 3, 1948.

volunteers, found that exercise tolerance as measured by currently used stress tests was markedly reduced for many weeks. Tilt-table studies on these subjects showed a tendency to fainting. During bedrest there occurred a loss of those circulatory adjustments necessary for the maintenance of erect posture. These changes were principally due to a diminution of tone of either muscles or vasculature of the lower extremities, with venous engorgement, stasis, edema, and increased capillary fragility. The patients in this series did not manifest these sequelae when ambulation was started.

The incidence of complications was negligible and in no instance attributable with any certainty to this plan of treatment. In the management of acute coronary thrombosis, pulmonary congestion and edema are common and serious complications. The most striking feature of this method of treatment is its effect on the pulmonary manifestations of cardiac failure. This was apparent in different ways. In those patients who were dyspenic or orthopneic or showed disseminated pulmonary rales, there was prompt improvement directly after they were placed in a chair. Also, in the four patients examined post mortem, the lungs were particularly free of edema. There was only one instance of pulmonary edema that developed after this method of treatment was instituted. This occurred in a woman on the 13th hospital day and was precipitated by a bout of paroxysmal rapid heart action. Satisfactory recovery took place with continued "chair" treatment.

Employing this treatment in alternate patients as a method of obtaining adequate control series was not feasible. The lack of such a selected control group precludes the statistical evaluation of our mortality figures. In some cases the physician and in others the patient's family refused to accept this method of treatment. The prevailing view is that patients with cardiac disease are expected to die in bed. If fatalities occur out of bed, the physician is held culpable. It should be emphasized that a significant number of the patients in this present study were seen in consultation practice; because of this, the series is weighted with gravely ill patients. It is of interest that during this limited experience there were 30 consecutive cases of coronary thrombosis in one of our public wards with only one fatality. The only basis of comparison that we have is the 138 consecutive patients with acute coronary disease treated with anticoagulants at the Peter Bent Brigham Hospital just prior to our study. The immediate mortality was 13.8%, compared with 8.3% for the "chair"-treated patients receiving anticoagulants. The small size of our present series and the lack of alternate controls prevents our attaching undue significance to this difference. We believe, however, that it has significance insofar as it helps dissipate the conception that the sitting position out of bed increases mortality among patients with acute coronary thrombosis.

The present trend in care of patients with coronary disease is a liberalization of the rigid restrictions of activity hitherto practiced. Our prime concern in this study has not been with activity of the patient, but rather with rest for the heart. A suggestion has been recently made that rest in bed be limited to two weeks, followed by a gradual increase in activity.[12] It should be borne in mind that most of the serious complications occur during the first two weeks, when the majority of fatalities occur and when pulmonary congestion begins to threaten. It is therefore during this very period that it is desirable to obtain the beneficial effects of the "chair" treatment.

A possible hazard of this treatment is that it may encourage an easy-going attitude toward the gravity of the process being treated. The fact that someone is in a chair evokes the feeling that the patient cannot be very ill. Such an approach might obviate some or all of the benefits achieved with this treatment. The tenacity with which bed rest has been promulgated for acute coronary thrombosis is no measure of its value, though the correct amount of true physiological rest is important. A proper conception of the principle of rest must embody an appreciation of the burdens on the heart resulting from the recumbent position and anxiety.

SUMMARY AND CONCLUSIONS

1. Physiological as well as clinical studies adduce evidence to support the view that strict rest in bed is injurious to the patient with congestive heart failure.

2. The sitting position in a chair with the feet dependent obviates some of the harm of strict bedrest.

3. This principle has been applied in the treatment of 81 patients with acute coronary thrombosis. These patients were kept in a chair varying and increasing portions of the day, beginning not later than the first week of the attack.

4. There were eight deaths, an over-all mortality of 9.9%. All other methods of treatment usually employed in coronary thrombosis were used. If only the patients who received anticoagulants are considered (72 patients), the mortality was only 8.3%. There were no complications attributable to the "armchair" treatment. The prompt improvement shown by some of those desperately ill with congestive heart failure after being placed in a chair was particularly impressive.

5. This method of treatment also appeared to have beneficial effects on the psychological state of the patient and facilitated the rehabilitation process.

270 Commonwealth Ave. (16) (Dr. Levine).

12. Irvin, C. W., Jr., and Burgess, A. M., Jr.: Abuse of Bed Rest in Treatment of Myocardial Infarction, New England J. Med. 243: 486, 1950.

Bad Habits in Children.—What are frequently thought of as "bad habits" in children, thumb-sucking, head-knocking, masturbation, nail biting, the ritualistic behavior in connection with getting to bed at night and many other such manifestations are more or less common at one or another time in the life of every child. These habits can be used negativistically because they are objectionable to the adults in charge and are for a certain period persistent actions which do not yield readily to external pressures. It is too seldom appreciated that behind this behavior there is anxiety which the child attempts to master in one of these ways, so as to maintain or restore his psychic equilibrium and comfort. These so-called "bad habits" cannot constructively be influenced except through the recognition of the underlying anxieties and through attempting to relieve them. Meanwhile, tolerance for the behavior and avoidance of attempts to suppress it should rest on an understanding of its unconscious purposeful nature.—Hunter P. Harris Jr., M.D., Some Aspects of Negativistic Phenomena, *Southern Medical Journal*, January, 1952.

ANOXIA IN MYOCARDIAL INFARCTION AND INDICATIONS FOR OXYGEN THERAPY

Craig W. Borden, M.D., Richard V. Ebert, M.D.
and
Russell H. Wilson, M.D., Minneapolis

Oxygen therapy has been widely employed in the management of patients with coronary thrombosis and myocardial infarction because it has been reputed to relieve pain.[1] Thrombosis of a coronary artery leads to necrosis of the myocardium in the area supplied by this vessel. Adjacent to the necrotic muscle is viable but ischemic muscle, which gives rise to pain and represents a threat to the patient in that it, too, may undergo necrosis or give rise to ventricular fibrillation. One of the logical aims of therapy of myocardial infarction is to provide a more adequate supply of oxygen to the ischemic muscle until collateral circulation can develop. The inhalation of oxygen may accomplish this aim by increasing the oxygen content of the arterial blood so that each unit of blood carries more oxygen to the anoxic tissue. Oxygen therapy is also used to relieve dyspnea in myocardial infarction and to prevent the general deleterious effect of anoxia.

Despite the universal practice of giving oxygen to patients with myocardial infarction, little information is available regarding the oxygen saturation of the hemoglobin of the arterial blood in this disease. It is apparent that information is needed as to the severity of anoxia if therapy is to be rational.

METHOD

Twenty-three men with acute myocardial infarction were studied as soon as possible after the diagnosis had been established by the classical criteria. Portable roentgenograms of the chest were made at the bedside the same day of the study and read for the presence or absence of pulmonary edema without knowledge of other data. After procaine hydrochloride infiltration, arterial blood samples were obtained anaerobically from the femoral artery. These samples were analyzed for oxygen content and capacity, carbon dioxide content and pH, with the use of methods previously described.[2] The pCO_2, was calculated from the nomogram of Singer and Hastings.[3] In calculation of the percentage of oxygen saturation of the hemoglobin, the following corrections were made: The oxygen content and oxygen capacity were corrected for oxygen dissolved in physical solution.[4] In calculation of the capacity, hemoglobin content was determined colorimetrically on blood samples before and after rotation in the tonometer to equilibrate with room air to correct for changes in red cell plasma ratio caused by drainage errors in glass vessels.[5] This correction in these data gave a decrease in the value of the determined oxygen capacity of 2.86%. When applied to data for normal men previously reported,[2] the mean normal oxygen saturation was 97.7%, a value similar to that reported by others.[6]

*Oxygen Saturation of the Hemoglobin of the Arterial Blood in Patients with Myocardial Infarction**

	No. Patients	Oxygen Saturation, % Average	Oxygen Saturation, % Range	pH Average	pH Range	pCO_2 Average	pCO_2 Range
Group I †.....	17	94.1	88.0-96.8	7.39	7.30-7.45	38.6	32.8-50.1
Group II ‡.....	6	80.8	61.4-92.4	7.39	7.35-7.45	41.6	31.5-58.5
Normal value.....		97.5		7.40		40	

* Values given represent mean and range for each group.
† Uncomplicated myocardial infarction.
‡ Myocardial infarction complicated by shock or pulmonary edema.

RESULTS

Seventeen of the 24 patients had no evidence of pulmonary edema, either clinically or roentgenographically, and no evidence of the shock syndrome (group I). Of the remaining six patients (group II), four had definite roentgenographic evidence of diffuse pulmonary edema and no symptoms of shock; one had pulmonary edema and the shock syndrome, and one had the shock syndrome without signs of pulmonary edema in the roentgenogram of the chest.

The results for each group are given in the accompanying table. The patients in group I showed only a slight reduction in oxygen saturation of arterial blood as compared with those in group II, who showed a marked reduction. The pH and pCO_2 in both groups were normal.

The dosage and time of administration of morphine were noted for each patient. Examination of the results revealed no evidence that morphine had depressed ventilation of the lungs. This may be because large amounts of morphine were not given and because morphine was given in the presence of pain.

From the Veterans Administration Hospital and the Department of Medicine, The Medical School, University of Minnesota.

Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

1. Rizer, R. I.: Oxygen in the Treatment of Coronary Occlusion: Preliminary Report, Minnesota Med. **12**: 506-507 (Aug.) 1929. Barach, A. L.: The Therapeutic Use of Oxygen in Heart Disease, Ann. Int. Med. **5**: 428-440 (Oct.) 1931. Bickel, G.: Le traitement de l'infarctus du myocarde, Paris méd. **23**: 388-397 (May 6) 1933. Kilgore, E. S.: The Treatment of Acute Coronary Occlusion, J. A. M. A. **100**: 315-318 (Feb. 4) 1933. Barach, A. L., and Levy, R. L.: Oxygen in the Treatment of Acute Coronary Occlusion, ibid. **103**: 1609-1693 (Dec. 1) 1934. Boland, E. W.: Oxygen in High Concentration for Relief of Pain in Coronary Thrombosis and Severe Angina Pectoris, ibid. **114**: 1512-1514 (April 20) 1940.

2. Wilson, R. H.; Borden, C. W.; Ebert, R. V., and Wells, H. S.: A Comparison of the Effect of Voluntary Hyperventilation in Normal Persons, Patients with Pulmonary Emphysema, and Patients with Cardiac Disease, J. Lab. & Clin. Med. **36**: 119-126 (July) 1950.

3. Singer, R. B., and Hastings, A. B.: An Improved Clinical Method for the Estimation of Disturbances of the Acid-Base Balance of Human Blood, Medicine **27**: 223-242 (May) 1948.

4. Sendroy, J., Jr.; Dillon, R. T., and Van Slyke, D. D.: Studies of Gas and Electrolyte Equilibria in Blood: The Solubility and Physical State of Uncombined Oxygen in Blood, J. Biol. Chem. **105**: 597-632 (June) 1934.

5. Roughton, F. J. W.; Darling, R. C., and Root, W. S.: Factors Affecting the Determination of Oxygen Capacity, Content and Pressure in Human Arterial Blood, Am. J. Physiol. **142**: 708-720 (Dec.) 1944.

6. Comroe, J. H., Jr., and Dripps, R. D.: The Physiological Basis for Oxygen Therapy, ed. 1, Springfield, Ill., Charles C Thomas, Publisher, 1950, p. 5.

COMMENT

The reduction in oxygen saturation of the hemoglobin of the arterial blood in myocardial infarction is caused in large part by pulmonary edema. The presence of edema fluid in the lungs interferes with diffusion of oxygen from the alveolar air to the blood in the pulmonary capillaries.[7] As a result, the oxygen tension in the blood leaving the lung is much lower than the alveolar oxygen tension. Carbon dioxide, on the other hand, diffuses much more readily through fluid, so that the carbon dioxide tension in the blood remains relatively normal.

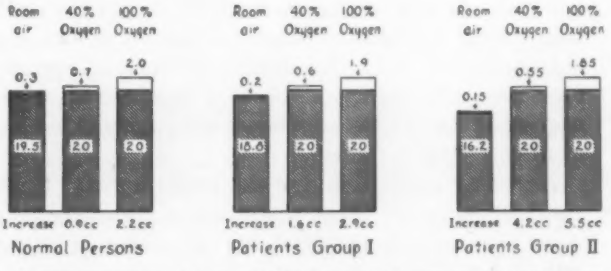
The purpose of this study was to define more accurately the indications for oxygen therapy in myocardial infarction. The results indicate that, in the patient without pulmonary edema or shock, the degree of anoxia is mild and of no importance to the body as a whole. The sole purpose of oxygen therapy in this group of patients would be the provision of greater amounts of oxygen to ischemic areas in the myocardium. On the other hand, in those patients with evidence of pulmonary edema or shock, the anoxia may be severe. Oxygen therapy is mandatory to combat the general effects of anoxia on the body as well as to supply additional oxygen to the myocardium. It is evident that the use of oxygen therapy is imperative when pulmonary edema or shock complicates myocardial infarction and is of doubtful value in the uncomplicated disease.

A consideration of the effect of oxygen therapy may be of value at this point. The amount of oxygen that can be added to the arterial blood by having the patient breathe oxygen instead of air is dependent on two factors: one is the oxygen saturation of the hemoglobin of the arterial blood, and the other is the concentration of the oxygen inhaled. The greater part of the oxygen in blood is chemically bound by hemoglobin. A small portion is in simple physical solution. This relationship is illustrated in the accompanying figure. In a normal human being breathing air, 19.5 cc. of oxygen is bound by hemoglobin in each 100 cc. of blood, and 0.3 cc. is in solution. The hemoglobin of the arterial blood is 97.5% saturated with oxygen. If this person now breathes a mixture of 40% oxygen and 60% nitrogen, the hemoglobin of the arterial blood will be completely saturated and the dissolved oxygen will be increased. The additional oxygen carried by each 100 cc. of arterial blood will be 0.9 cc. Of this amount, 0.5 cc. will represent additional oxygen carried by the hemoglobin. If 100% oxygen is breathed, the amount of oxygen chemically bound by the hemoglobin is the same, but an additional 1.3 cc. of oxygen can be physically dissolved in each 100 cc. of blood. Thus the maximum amount of oxygen that can be added to the arterial blood of a normal person by breathing 100% oxygen is 2.2 cc. for each 100 cc. of blood. On the other hand, if a patient is anoxic and the hemoglobin of the arterial blood is unsaturated, much more oxygen can be added to the arterial blood. The average oxygen saturation of the hemoglobin of the arterial blood of the patients in group II was 80.8%. Inhalation of 40% oxygen would add 4.2 cc. of oxygen to each 100 cc. of arterial blood, of which 3.8 cc. would be chemically bound by hemoglobin. Breathing 100% oxygen would add 5.5 cc. of oxygen to each 100 cc. of blood. The corresponding figures for the patients in group I would be a 1.6 cc. increase by inhalation of 40% oxygen and a 2.9 cc. increase by inhalation of 100% oxygen.

From the above consideration it is evident that relatively little additional oxygen is added to the blood of patients with uncomplicated myocardial infarction by administration of oxygen in a concentration of 40%. Nearly twice the amount of oxygen can be added to the blood if the patient inhales 100% oxygen. Hence, if oxygen is to be administered at all to patients with uncomplicated myocardial infarction, it would appear preferable to utilize a mask rather than an oxygen tent. Under optimum conditions a concentration of 40% oxygen can be achieved in an oxygen tent.[8] If care is not exercised, the concentration is 25% to 30%. With the proper use of the BLB mask, a concentration of oxygen of 92% can be obtained in the alveolar air.[9]

SUMMARY

The oxygen saturation of the hemoglobin of the arterial blood was studied in a group of 23 patients with myocardial infarction. The mean value in those patients without pulmonary edema or shock was 94.1%, and the mean value in those patients with pulmonary edema or shock was 80.8%.



Oxygen content of 100 cc. of blood in normal persons and both groups of patients breathing room air, 40% oxygen and 100% oxygen. Shaded area represents oxygen chemically bound by hemoglobin. Clear area represents oxygen in physical solution.

On the basis of these findings it is apparent that the use of oxygen therapy is mandatory in those patients with pulmonary edema or shock and of questionable value in the patients with uncomplicated myocardial infarction. If oxygen is used in the latter group, it should preferably be given in high concentration by mask rather than in the lower concentration obtained by use of an oxygen tent.

7. Meakins, J. C., and Davies, H. W.: Respiratory Function in Disease, Edinburgh and London, Oliver & Boyd, Ltd., 1925, p. 300.

8. Wolff, M. H.: Oxygen Concentration in Oxygen Tents, U. S. Nav. M. Bull. **44**: 988-990 (May) 1945.

9. Boothby, W. M.; Lovelace, W. R., Jr., and Uihlein, A.: The B. L. B. Oxygen Inhalation Apparatus: Improvements in Design and Efficiency by Studies on Oxygen Percentages in Alveolar Air, Proc. Staff Meet., Mayo Clin. **15**: 194-206 (March 27) 1940.

Local Anesthesia.—Epinephrine in 1:200,000 dilution will greatly prolong (triple or quadruple) the action of local anesthetic agents. Use 0.5 cc. of a 1:1,000 solution of epinephrine to 100 cc. of 1 per cent procaine hydrochloride. However, epinephrine or other vasoconstrictor agent is not to be used with local anesthetic agents in surgery of the fingers, toes, ears, nose, penis or scrotum. Sloughs may be caused if this is done. Epinephrine is not to be added to local anesthetics if their action is to be supplemented with cyclopropane, chloroform or ethyl chloride.—Henry K. Beecher, M.D., Early Care of the Seriously Wounded Man, Springfield, Charles C Thomas, Publisher, 1952.

HISTORY OF THE TREATMENT OF CORONARY HEART DISEASE

FRANK BILLINGS LECTURE

N. C. Gilbert, M.D., Chicago

It was not my good fortune to have been one of Dr. Billings' students, but I was fortunate in my association with him in various medical affairs. He was always interested and interesting. I never failed to be impressed by his store of practical wisdom, his sense of relative values, and his quick recognition of the essential details of any matter under discussion.

Dr. Billings contributed a great deal to American medicine. The American Medical Association is greatly indebted to him for his leadership in much of the best that this organization represents. Together with hundreds of others, I owe him a great deal. He was a strong man in every way and a leader and teacher of men.

The story of angina pectoris as an expression of coronary disease long precedes Heberden's "Some Account of a Disorder of the Breast," which established it as a clinical entity. But it is with Heberden [1] that any account of treatment begins. It was over 100 years after Heberden that coronary occlusion was differentiated as a distinct entity, the basis of which is coronary insufficiency. George Dock,[2] in another Billings lecture, has given an account of the history of coronary disease and of coronary occlusion; I shall confine myself largely to the treatment of coronary disease.

Heberden's description of the indications for therapy are quite as we see them now. He was impressed by the symptoms suggesting "ills justly attributed to the disturbed function of the nerves." For generations afterward, the nervous aspects of angina pectoris have continued to be given weight. "With respect to the treatment of this complaint," wrote Heberden, "I have little or nothing to advance. Quiet and warmth and spirituous liquors help to restore patients who are nearly exhausted and to dispel the effects of a fit which does not soon wear off. Opium taken at bedtime will prevent the attacks at night. Bleeding, vomits, and purging appear to me to be improper." But in his Commentaries [1] he added: "Wine or spirits mixed with water have gradually led on several to be sots and have ruined more constitutions than ever were hurt by small beer from its first invention."

Heberden's conservative methods were not always adhered to. In 1795 Samuel Black [3] reported a case in which he used "issues," "blisters," and, as he says, "a variety of medicines adapted to the indications were prescribed, such as antimonial and mercurial alteratives, the volatile tincture of guaiac with antimonial wine, laxatives, etc." He tells of a contemporary physician of eminence and much experience, who gave his patient tar water "to the amount of a jill three times a day." This was "uniformly observed to excite a considerable diuresis." For the spasmodic nature of the attacks, Black tried a variety of remedies, such as "the Peruvian bark, the fetid gums, camphor, and the extractum cicutae (hemlock) to the amount of 20 grains twice a day in which dose it excited headache and tremors. For the relief of the paroxysms, when present, I had recourse to the vitriolic ether and the tincture thebaica in peppermint water. . . . Notwithstanding such varied treatment, the effects of the remedies employed were far from corresponding with my earnest wishes." Dr. Haygarth,[4] prescribed for one of his patients an anodyne draft to be repeated every two or three hours if the pain did not abate. "A blister was also applied to the part." The patient was not better the next morning, so 12 oz. of blood were taken, and a "draught of Camph., gr., x. and Nitr., gr. xv., was given every 4 to 6 hours. And he was directed to drink very plentifully of acidulated diluting liquids. After using these he found immediate relief, and by continuing this method with a dose of Rochelle salts every second or third morning, his pain and fever in a few days entirely ceased. He gradually recovered."

Jenner [5] apparently had little faith in treatment, for in a body dissected by Paytherus, he had seen a thickening of the coronary arteries and what is now regarded as coronary thrombosis. In a letter to Heberden concerning anginal attacks, from which his friend John Hunter was suffering, he stated, "Should it be admitted that this [coronary changes] is the cause of the disease, I fear the medical world may seek in vain for a remedy." Hence, he avoided discussing his findings and his views for fear of alarming his good friend Hunter.

Parry [6] was certain that the cause of angina pectoris lay in the coronary arteries, "either ossified or cartilaginous," and advised very conservative treatment. He considered that "mischief might be done by some drugs." "On the whole," he said, "it seems that during the paroxysm stimulants can be safely used only so far as they are required to get the gas off the stomach." Allan Burns,[7] in 1809, was a follower of Parry. He might, during a paroxysm, "detract a few ounces of blood." "Our decided object must be to allow the exhausted heart to slowly recover its lost energy." He might "hold some ammonia

---

Read before the Section on Internal Medicine at the One-Hundredth Annual Session of the American Medical Association, Atlantic City, June 13, 1951.

Each year, at this time, it is the custom that an address be delivered before this Section, in honor of Dr. Frank Billings, one of the most distinguished of its members.

1. Heberden, W.: Some Account of a Disorder of the Breast, M. Tr. Coll. Physicians **2**: 59, 1786. Commentaries on the History and Cure of Diseases, Boston, Wells and Lilly, 1818.

2. Dock, G.: Historical Notes on Coronary Occlusion: From Heberden to Osler; Billings Lecture, J. A. M. A. **213**: 563 (Aug. 12) 1939.

3. Black, S.: A Case of Angina Pectoris, with Remarks, Mem. M. Soc. London **4**: 261, 1795.

4. Haygarth, J.: A Case of Angina Pectoris, with an Attempt to Investigate the Cause of the Disease by Dissection, and a Hint Suggested Concerning the Method of Cure, M. Tr. Coll. Physicians, **3**: 37, 1785.

5. Jenner, E.: Letter to Dr. Heberden, The Life of Edward Jenner, M.D., with Illustrations of His Doctrines and Selections from His Correspondence, in Baron, J.: London, Henry Colburn, 1838, vol. 1, p. 39-40.

6. Parry, C. H.: quoted by Burns, A.: Observations on Some of the Most Frequent and Important Diseases of the Heart, Edinburgh, Bryce, 1809.

7. Burns, A.: Observations on Some of the Most Frequent and Important Diseases of the Heart, Edinburgh, Bryce, 1809.

over the nose," or give some aromatic powder to "aid in the expulsion of wind from the stomach."

Laennec [8] did not admit that coronary disease had anything to do with angina, although the two might coincide. He considered angina pectoris a neuralgia or rheumatism. He used two strongly magnetized steel plates anteriorly and posteriorly over the cardiac area. "The method is not infallible," he stated, "but it succeeded better than others." Hope [9] observed that "they who witnessed the application, did not, in general, form so favorable an opinion as the author himself." If Laennec had stopped there, the advantages of so harmless a treatment would have been obvious, but, when Laennec's magnetic plates did not succeed, he used blisters, sinapisms, venesection or leeches, wild laurel, digitalis, and the old reliable fetid gums. Then, as now, physicians found it just as hard to keep their hands off the medicine bottle as for a toper to keep his hands off the liquor bottle, I wish that I might continue to tell more of the early treatment of angina pectoris. One can not read the early records without a great sense of admiration for "our fathers of old," who were so sincere in their efforts and who had so little to do with. We who follow, have so much more to work with should feel very humble. Modern students could also learn much from the accounts of Gairdner,[10] Gibson,[11] Allbutt,[12] and the Osler monograph.[13]

For the next several decades angina pectoris was treated symptomatically by each physician according to his own lights. No matter what the basic diagnosis, whether spasm of the heart, diaphragmatic gout, or neuralgia, the treatment varied little—rest, diet, avoidance of undue effort, avoidance of gastric disturbances and relief of flatulence, spirituous liquors, sinapisms, pediluviums, spirits of nitrous ether, chloroform inhalations, Hoffmann's anodyne, strychnine, bromides, quinine, and digitalis. Treatment by bleeding also persisted for a long time. The importance of indigestion and of flatulence was well recognized. Carminatives were given constantly. Between the attacks, colchicum and tonics, especially iron, were used. The reports of successful use of arsenic were such that one almost becomes converted to its use. Trousseau [14] used atropine, but oftener belladonna, with or without sodium bicarbonate or carbonate. In this he was influenced by Bretonneau, "whose practical sense had not been stultified by the most extensive chemical knowledge."

In 1867 was made the first constructive contribution based upon what is now regarded as rational therapy, even though at the time the effect on the coronary vessels was not recognized. Dr. T. Lawder Brunton,[15] just finishing his medical studies, and a resident physician in the Edinburgh Royal Infirmary, was impressed by the effect of amyl nitrite in lowering blood pressure. He tried this drug on a patient with anginal attacks, and, after the patient had inhaled the fumes of from 5 to 10 drops, Dr. Brunton stated that his hopes were fulfilled. In 1879, there appeared serially in the *Lancet* a description of the effect of nitroglycerin by Dr. William Murrell.[16] This work further advanced the therapy of angina pectoris, although, at the time, the effect was ascribed to the relief of arterial tension. Dr. W. H. Madden,[17] in 1872, gave a report of his personal experience with amyl nitrite. He had supposed that its use was only for those whose faces were pallid during an attack, and because his face was flushed, he neglected using this drug. When he at last gave it a trial, the effect, he wrote, "was truly wonderful." It not only stopped each attack, but the attacks became less frequent. In describing his relief he said. "The presence of intense pain is not conducive to the exercise of calm, philosophic analysis, and I can only tell you what I felt." It was not until the observations of Sir Thomas Lewis [18] that it was shown that the relief of pain was due to the increased coronary flow and not necessarily to the decrease in peripheral resistance.

The diuretic effect of the xanthines had been recognized for at least 10 years, when, in 1895, Askanazy [19] reported improvement in cardiac asthma and in angina pectoris with the administration of theobromine sodium salicylate. He had observed this by chance when using that salt for its diuretic effect. This attracted little attention until 1902, when Breuer [20] wrote an article on angina pectoris. He wrote that, after 5 years' experience with theobromine, he could confirm Askanazy's results and that he regarded the recommendation of Askanazy as "one of the most praiseworthy therapeutic attainments of the last ten years." It was not long before the pharmacologists, starting with Hedbom [21] and Loeb,[22] followed by many others, showed that this series of drugs had a direct vasodilator effect on the coronary arteries, increasing the flow through the arteries and producing a more prolonged effect than the nitrites. A variety of other preparations have followed, with various advantages and disadvantages, up to the khellin preparations of recent date.

When symptoms are the result of insufficient blood flow to the heart muscle, it would seem rational to use agents that tend to increase that flow by direct action on the vessel wall, relaxing the vessel, without at the same time introducing other variables. While the use of coronary vasodilator drugs is common now, there are still those who profess not to obtain good clinical results.

---

8. Laennec, R. T. H.: A Treatise on the Diseases of the Chest and on Mediate Auscultation, New York, S. Wood & Son, 1830, p. 702.

9. Hope, J.: A Treatise on the Diseases of the Heart and Great Vessels, London, Kidd, 1932, p. 466.

10. Gairdner, W. T.: Angina Pectoris and Sudden Death in Reynolds, J. R.: A System of Medicine, Philadelphia, Henry C. Lea's Son Co., 1880, vol. 2, p. 665.

11. Gibson, G. A.: Diseases of the Heart and Aorta, Edinburgh and London, Y. J. Pentland, 1898. Nervous Affections of the Heart, Edinburgh M. J. **3**: 324, 1903.

12. Allbutt, T. C.: Diseases of the Arteries Including Angina Pectoris, New York, The Macmillan Company, 1915.

13. Osler, W.: Lectures on Angina Pectoris and Allied States, New York, D. Appleton & Company, 1897.

14. Trousseau, A.: Lectures on Clinical Medicine Delivered at the Hôtel-Dieu, Paris, Philadelphia, Lindsay & Blakiston, 1867.

15. Brunton, T. L.: On the Use of Nitrite of Amyl in Angina Pectoris, Lancet **2**: 97, 1867.

16. Murrell, W.: Nitroglycerin as a Remedy for Angina Pectoris, Lancet **1**: 80; 113; 151; 225, 1879.

17. Madden, W. H.: Nitrite of Amyl in Angina Pectoris, Practitioner **9**: 331, 1872.

18. Lewis, T.: Material Relating to Coarctation of the Aorta of the Adult Type, Heart **16**: 205, 1933.

19.. Askanazy, S.: Klinisches über Diuretin, Deutsches Arch. klin. Med. **56**: 209, 1895.

20. Breuer, R.: Zur Therapie und Pathogenese der Stenokardie und verwandter Zustande, München. med. Wchnschr. **49**: 1604, 1902.

21. Hedbom, C.: Ueber Die Einwirkung gewisser Pflamzengifte Skandinav. Arch. f. Physiol. **9**: 1, 1899.

22. Loeb, O.: Uber die Beeinflussung des Koronarkreislaufs durch einige Gifte, Arch. f. exper. Path. u. Pharmakol. **5**: 64, 1904.

There are, in coronary disease, a great many interacting variables, ranging from irreversible anatomic changes in the vessel walls and in the heart itself to those of vasomotor influence. No one agent, no matter how effective it might be in any one particular, can cure coronary disease in every instance.

Jenner, Parry, and Allan Burns believed that angina pectoris depended on changes in the coronary arteries. In 1842, John Erichsen [23] showed that ligation of a coronary artery stopped the heart and that increasing the coronary flow strengthened the heartbeat. Dr. Marshall Hall,[24] in the same year, said that ossification of the coronary arteries was a common cause of angina pectoris and death. Nevertheless, it was difficult to convince many that deficiency in coronary flow was the basis of angina pectoris. If this were true, asked the dissenters, why did the symptoms come and go, as they so often did, and why was the health perfect between attacks? Why did eating, indigestion, flatulence, walking against the wind, or breathing cold air bring on attacks; and why did the attacks disappear in some patients? Why did not all patients show sclerosed arteries at autopsy? Why did some persons live to a very comfortable old age, with very hard arteries? For these reasons Laennec and Trousseau and many others after them called the attacks neuralgia. Hanfield Jones,[25] as late as 1870, and Anstie,[26] in 1871, included angina pectoris as a neuralgia in their respective texts. Austin Flint [27] considered it a neuralgia, and it is classified as such as late as the 1896 edition of his book.

Landois,[28] in 1866, postulated a reflex angina pectoris from visceral sources. Allbutt held that the theory of vasomotor angina was merely "an argumentative resource, first supplied, I think, by the ingenuity of von Neusser." He did not consider that coronary arteries were the basis of angina, and he was supported by many others among them Wenchebach and Vaquez. One would suppose that clinical observations combined with a little reflection would have suggested that vasomotor narrowing of the coronary arteries would cause the same symptoms as did anatomic narrowing and would have reconciled the discrepancies in the clinical picture. Experimental proof of a reflex decrease in coronary flow was supplied by the experiments of von Bergmann [29] and his associates in 1932. They showed that reflexes decreasing the coronary flow could be produced by distention of the stomach or the esophagus, and especially of the stomach at the hiatal orifice. This work has been repeated and confirmed by others, who showed that distention of the free abdominal cavity with air also caused a reflex decrease in coronary flow. These reflexes were abolished by vagal section or by atropine. Similar reflexes have been produced by distention of the gallbladder or of the cystic duct. These reflexes were also abolished by atropine. The results have been substantiated by clinical experiments, and we see the evidence daily in our offices and in our wards. It has also been shown that stimulation of the nasal mucous membrane by an irritating vapor or cold could decrease the coronary flow. Clinical experience confirms the laboratory experiments.

We can now see that the importance that Allbutt placed upon vagus stimulus was well deserved, even though misinterpreted. Two other contributions, which have not received the clinical attention they deserve, were made by Rein [30] in 1931 and Greene [31] in 1936. Rein showed the presence of a constant vagal tone in the coronary arteries, explaining the effect of atropine on the coronary flow. Greene showed the vagus nerve to be the sole pathway of efferent coronary vasoconstrictor fibers. Workers at the Banting Institute [32] showed that stimulation of the vagus nerve, either centrally by drugs or peripherally by electrical stimulation, caused degenerative changes in the heart muscle because of constricted coronary arteries and decreased blood flow to the heart muscle. The clinical importance of this experiment lies not only in the vasoconstrictor effect of the vagus upon the coronary arteries but in the effect of repeated episodes of decreased coronary flow in producing permanent degenerative changes in the heart muscle.

Treatment, in general, from the time of Brunton, evolved very slowly. It has always been difficult for the physician to decide what the abnormal conditions are and which agents are necessary to meet those conditions, or, if treatment is not possible, to do nothing. After Brunton, the nitrites were used almost universally, even by those who considered that they were treating a cardiac neuralgia. The articles of Askanazy and Breuer received little attention, rational as they were. The influence of tradition continued strong, as it does today. After Huchard,[33] the iodides were used very generally. Osler used them, but not so enthusiastically as Huchard. Osler occasionally used colchicum and chloroform inhalations for the relief of the paroxysms. In a later text, he recommended theobromine. In his monograph on angina pectoris, Mackenzie [34] wrote, "Digitalis and drugs allied to it have long been recognized as having an effect upon the heart, but the more these drugs are studied, the clearer it becomes that it is only in *certain definite* conditions that they act, and their sphere of influence is very limited." Gibson writes of digitalis, "It is not of conspicuous utility." Digitalis is still used widely in coronary disease, more because of tradition than for definite indications for its use. The survival of a patient after receiv-

23. Erichsen, J. E.: On the Influence of the Coronary Circulation on the Action of the Heart, London M. Gaz. **30**: 561, 1842.

24. Hall, M.: On the Mutual Relations Between Anatomy, Physiology, Pathology, and Therapeutics, and the Practice of Medicine; Being the Gulstonian Lectures for 1842, London, H. Baillière, 1842.

25. Jones, C. H.: Studies on Functional Nervous Disorders, ed. 2, London, J. Churchill & Sons, 1870.

26. Anstie, F. E.: Neuralgia and the Diseases that Resemble it, New York, The Macmillan Company, 1871.

27. Flint, A.: A Treatise on the Principles and Practice of Medicine: Designed for the Use of Practitioners and Students of Medicine, ed. 7, Philadelphia, Lea Brothers & Co., 1894.

28. Landois, L.: Der Symptomencomplex "Angina Pectoris" Physiologisch Analysiert, nebst Grundlinien einer rationellen Therapie, Correspondenz-Blatt der deutschen Gesellschaft für Psychiatrie und gerichtliche Psychologie **13**: 2, 1866.

29. von Bergmann, G.: Das "epiphrenale Syndrom," seine Beziehung zur Angina Pectoris und zum Kardiospasmus, Deutsche med. Wchnschr. **58**: 605, 1932.

30. Rein, H.: Die Physiologie der Herz-Kranz-Gefässe, Ztschr. f. Biol. **92**: 101; 115, 1931.

31. Greene, C. W.: Analysis of the Relations of the Coronary Constrictor and Dilator Nerves in the Cervical Vagosympathetic of the Dog, Am. Heart J. **11**: 592, 1936.

32. Hall, G. E.; Ettinger, E. H., and Banting, F. G.: An Experimental Production of Coronary Thrombosis and Myocardial Failure, Canad. M. A. J. **34**: 9, 1936. Ettinger, E. H.; Hall, G. E., and Banting, F. G.: Effect of Repeated and Prolonged Stimulation of the Vagus Nerve in the Dog, ibid. **35**: 27, 1936. Manning, G. W.; Hall, G. E., and Banting, F. G.: Vagus Stimulation and the Production of Myocardial Damage, ibid. **37**: 314, 1937.

33. Huchard, H.: Maladies du Coeur et des vaisseaux, artério-sclérose, aortites, cardiopathies artérielles: Angines de poitrine, etc., Paris, O. Dorn, 1899, vol. 2.

34. Mackenzie, J.: Angina Pectoris, London, Hodder & Stoughton, 1923.

ing any medication is not necessarily an index of its beneficial influence. Allbutt, who was most scornful of the "coronarians," used the nitrites for the acute attack and used theobromine and atropine in routine treatments. He used atropine and plenty of it for the purpose of blocking any vagal inhibitory action upon the heart and avoiding the vagal cardiac standstill. It is difficult to see how he could read his own book and not feel certain of the influence of the coronaries in angina pectoris. Verdon,[35] who considered that the pain of angina pectoris had its origin in a hollow viscus whose wall was under tension, paid a great deal of attention to dietary habits, as did almost all of the authors. He was so concerned with the importance of gas that he recommended the use of a stomach tube and tauhgt his patients to belch. He also used the conventional remedies, including aspirin and atropine. Lewis [36] used nitrites for the acute attack, and then theobromine, ammonium bromide, or phenobarbital.

Until our own century, coronary occlusion and temporary coronary insufficiency were not differentiated clinically. It was thought that the disproportion between coronary flow and myocardial needs might be due to actual occlusion, to too great demands upon anatomically narrowed vessels, to vessels constricted by vasospasm, or to a decrease in coronary flow due to a fall in systemic pressure. All through the history of coronary disease, it was evident from clinical and pathological studies that many cases of coronary occlusion were being diagnosed as angina pectoris. Beginning in the 1880's, some attempts at differentiation became apparent. In 1896 George Dock [37] published the reports of four autopsy cases of coronary occlusion due to thrombosis, in one of which the autopsy findings were anticipated during life. It seems strange that this report did not have more clinical influence than it did, but physicians then and now have a way of walking down the narrow path of their own preconceived convictions, looking neither to the right nor to the left, and not letting their eyes stray from the straight path before them. A glance at either side might often be of interest and show something worth stopping to examine more closely. Herrick's [38] article appeared in 1912, clearly differentiating coronary occlusion as a distinct entity. This article carried a tremendous impact upon clinical thought and practice, although the effect of the impact was slow. Someone once asked Newton how he had made his discoveries; his reply was, "by thinking of them." Harvey's work was based not so much upon his own experiments and those of others but upon rational reasoning concerning those experiments. The results did not come from laboratory glassware or animal experiments but from thoughtful consideration of the significance of what he already knew. This is true of the work of Herrick and before him, of Dock. Herrick himself had little to say regarding treatment and held gloomy views as to the future outlook of the patient. Lewis treated the pain symptomatically but was careful to prescribe two months of complete rest for the crippled heart.

The observations of Manning, McEachern, and Hall [39] showed a rational way to help decrease the mortality and the amount of residual damage. They showed that when a coronary artery is occluded, there is a reflex vasoconstriction of all the coronary arteries, decreasing the blood supply to all the heart muscle and, in consequence, decreasing the blood flow through the anastomosing vessels to the infarcted area. This reflex vasoconstriction was obviated by vagal section or atropine, and the mortality of the experimental animal was greatly reduced. LeRoy [40] repeated the work with similar results and lowered the experimental mortality still further by using a coronary vasodilator agent combined with atropine. Clinical treatment along similar lines has been found effective.

With the advent of anticoagulant therapy, advocated by Wright and others,[41] the mortality resulting from coronary thrombosis in the cases reported by them was materially reduced, as was the proportion of peripheral vascular accidents. There is no doubt as to the favorable effect of these drugs in cases of coronary thrombosis, but there is room for doubt as to just which property of these agents produced favorable effects. It is difficult to understand how any anticoagulant action could have an effect on an occlusion already present and firmly established or on the resultant established anemic infarct. There is no evidence that thrombi propagate. Viscosity is not altered. We do not know that there is any increase in the coagulability of the blood before the attack, even though there may be secondary effects. We do know that a large number of coronary occlusions are due not to a primary thrombosis but to subintimal hemorrhages, with, in many cases, a superimposed thrombus; however, these same anticoagulant agents do possess a vasodilator quality, which might offer a more valid reason for their favorable effect. This is especially true of bishydroxycoumarin (dicumarol®), which has a pronounced and prolonged vasodilator effect.

When an occlusion of a coronary artery occurs, with the resulting myocardial infarct, there is an immediate effort on the part of the body to restore the circulation in the anemic area. Any agent that will dilate the coronary vessels and increase their flow should increase the blood flow through the anastomosing vessels. This should help to meet the conditions imposed, especially if used after an initial dose of atropine, which relieves the reflex vasoconstrictor spasm. Barnum,[42] in a small but personally supervised series of 118 cases, using a coronary vasodilator drug that did not possess any anticoagulant action, had a low mortality rate and an equally low incidence of peripheral vascular accidents. The coronary vasodilator drug was used at once and administration was continued indefinitely. No other medication except

35. Verdon, H. W.: Angina Pectoris, London, Bailliere, Tindall & Cox, 1920.
36. Lewis, T.: Diseases of the Heart Described for Practitioners and Students, New York, The Macmillan Company, 1933.
37. Doch, G.: Some Notes on Coronary Arteries, M. & Surg. Reporter, **75**: 1, 1896.
38. Herrick, J. B.: Clinical Features of Sudden Obstruction of the Coronary Arteries, J. A. M. A. **59**: 2015 (Dec. 7) 1912.
39. Manning, G. W.; McEachern, C. G., and Hall, G. E.: Reflex Coronary Artery Spasm Following Sudden Occlusion of Other Coronary Branches, Arch. Int. Med. **64**: 661 (Oct.) 1939.
40. LeRoy, G. V., and others: The Influence of Xanthine Drugs and Atropine on the Mortality Rate After Experimental Occlusion of a Coronary Artery, Am. Heart J. **23**: 637, 1942.
41. Wright, I. S.; Marple, C. D., and Beck, B. F.: Anticoagulant Therapy of Coronary Thrombosis with Myocardial Infarction, J. A. M. A. **138**: 1074 (Dec. 11) 1948.
42. Barnum, D. R., and others: Coronary Thrombosis Treated with Xanthines: Experience from the Standpoint of Mortality and Incidence of Thromboembolic Complications, Quart. Bull. Northwestern Univ. M. School **24**: 6, 1950.

the atropine was used initially. Belladonna and phenobarbital were used later; a narcotic was given at first, if necessary, and oxygen for a short time, and, of course, bed rest. There was no other variable. This was a small series, but it is difficult to gather even such a small series in which some well-intentioned person has not introduced a variable of some kind at some time.

Hochrein [43] in 1937 wrote an article on the prevention of coronary occlusion, which has not received the attention it so well merits. He said that, while there are many cases of coronary occlusion with no antecedent history, there is a greater proportion of cases in which the previous history should lead one to suspect that at some time a coronary occlusion might occur. He speaks of emotional incidents as a precipitating cause and calls attention to the part that vascular spasm of the coronary arteries may play in the production of coronary occlusion.

A reduction in coronary flow due to various reflex causes has already been mentioned as well as its experimental confirmation. In reviewing the histories of cases of coronary occlusion, one frequently sees the same pattern of events that is found in cases of angina, the same incidence of the effects of fatigue and emotional disturbance, and the same background for reflex vasomotor incidents. Banting and his associates in their experiments produced intravascular thrombi in the coronary by vagus stimulation. Nalefski has produced infarcts of the heart muscle by the vasoconstrictor action of barium chloride, although no thrombi have been shown as yet.

If blood were so eager to clot that an anticoagulant was necessary, one might expect that the clotting would choose some nice, quiet, placid pool of venous blood instead of a very busy spot in the coronary artery. But, if the intima of a coronary artery were damaged this would predispose to the deposition of platelets in this area and the formation of a white and red thrombus, even in the presence of blood of normal or low coagulability. The walls of the vessels in coronary occlusion are usually already damaged, but a thrombus does not always occur in the most severely damaged vessels, and spasms may occur more readily in a less severely damaged vessel. A vasoconstrictor episode, by compressing the media and decreasing the blood supply to the intima, could readily cause intimal damage in an already damaged vessel wall. There is reason to think that it might also increase the wettability of a normal vessel wall, as in Banting's experiment.

There is yet much to be learned in regard to coronary disease, in part from the laboratory but largely from clinical observations over long periods of time, and in learning to use what we already know. To quote Dr. Dock in his 1895 article: "It must therefore remain for the physicians in general practice who have an oversight of patients over long periods and who at the same time have the same kind of scientific zeal that influenced a Jenner, to give the final touches to the picture of coronary disease."

104 S. Michigan Blvd.

43. Hochrein, M.: Können für die Entstehung des Myokardinfarktes verhuten, München. med. Wchnschr. **80**: 1613, 1933. Der Myokardinfarkt: Erkinnung, Behandlung und Verhütung, Dresden und Leipzig, T. Steinkopff, 1937.

DETECTION AND TREATMENT OF NUTRITIVE FAILURE IN CHILDREN

RECENT OBSERVATIONS

Tom D. Spies, M.D., Samuel Dreizen, D.D.S., George S. Parker, D.D.S.
and
Donald J. Silberman, M.D., Birmingham, Ala.

The diagnostic term "nutritive failure" means the same to us whether applied to children or adults. This term perhaps means different things to different physicians. It is used in our clinic to describe a variable clinical picture and does not have a specific physiological connotation. It does not indicate why the nutrition has failed but simply that it has failed. The orderly process of growth can be retarded in many ways by inadequate or improper nutrition. To evaluate these effects, we need to know as much as possible about the growing process of the child.

The growth of the body goes on under our eyes every day. The phenomenon is extremely delicate; yet, because it is so familiar, we think little of it. How do we develop cell by cell from the food we eat, the water we drink, and the air we breathe? How is the plan for growth, which has been defined as an increase in size and cell complexity, carried out? At birth the infant is roly-poly, with short arms and legs, and with a big head and abdomen. Then there is a progressive alteration in form and proportion. The chest develops rapidly and the head slowly. Adolescence comes, and the body shoots up with great speed. Finally, the ossification of the bones is completed, and we have maturity. If all has gone well, the results are most gratifying. What regulates the relationship between the various body cells? Is it the ductless glands? Do the thyroid, the pituitary, and the gonads have a vote? Can they always agree? If the pituitary is overactive, the result will be a large, tall person with long arms and legs. If the thyroid is underactive, the result will be a short person with delicate arms and legs. The gonads, when hyperactive, seem to produce an abundance of hair, strong bones, and a deep voice. What tends to equalize these

Dr. Dreizen is a Clayton Foundation Fellow, and Drs. Parker and Silberman are Williams-Waterman Fellows.

Read before the Section on Pediatrics at the One-Hundredth Annual Session of the American Medical Association, Atlantic City, June 14, 1951.

From the Department of Nutrition and Metabolism, Northwestern University Medical School, Chicago, and the Nutrition Clinic, Hillman Hospital, Birmingham.

This study was aided by grants from the Birmingham Citizens Committee Fund, the American Dry Milk Institute, Inc., and the Armour Research Laboratories.

activators, and how does the right result come about? That has been one of our major problems in the Nutrition Clinic of the Hillman Hospital for many years. We have learned that there is an interrelationship between vitamins, minerals, amino acids, and hormones. This interrelationship is so delicate that it makes one very humble in his approach to this problem.

In many instances we do not know enough to prescribe properly. We do know enough to be certain that the average patient does not understand dietary advice when given loosely in such terms as "eat sufficient meat, eggs, fresh vegetables, and fruit." How can we advise the parent intelligently until we have more information?

FOLIC ACID

The representative history given in case 1 shows a close physiological relationship between failure of hemopoiesis in the mother and failure of hemopoiesis in the nursing infant when they both are deficient in folic acid. Zuelzer[1] has shown that folic acid promotes hemopoiesis in infants with megaloblastic anemia. Angulo and one of us (T. D. S.)[2] have shown that the diets of patients with sprue and of some women with the macrocytic anemia associated with pregnancy were low in folic acid content. The latter (T.D.S.)[3] has described the close relationship between the macrocytic anemia of pregnancy and the anemia of infancy and early childhood and has pointed out that these anemias can be prevented by treatment of the mother in the late stages of pregnancy with substances like folic acid or foods containing adequate amounts of folic acid. In view of these facts, it follows that the pregnant woman should have ample folic acid in her diet or should be given synthetic folic acid or dried yeast, which contains folic acid, in order to prevent nutritional megaloblastic anemia in the mother and in the infant.

We have studied eight pregnant women who took mixed vitamin therapy under the impression that they were getting the vitamins they needed during pregnancy (unpublished observations). These vitamin preparations did not contain folic acid; since the diets of the women were low in folic acid, typical macrocytic anemia of pregnancy developed. It goes without saying that, if these patients had had sufficient synthetic folic acid and dried yeast, which contains folic acid, the anemia would not have developed.

CASE 1.—C. G., a 27-year-old woman, and her 8-week-old female infant were brought to the hospital in an ambulance. For several days the mother had been in a semicomatose state, and the infant had been very ill. The husband became alarmed about their condition and brought them both to the hospital. From the husband, and later from his wife, the following history was obtained.

The infant was the fifth child, the oldest being 6 years of age. The other four children were somewhat underweight but seemed to be in good health. During the first four or five months of each pregnancy the mother lost her appetite, was nauseated, and frequently vomited. Because of nausea, she ate only small amounts of foods on which she had subsisted all her life—bread, cornmeal, rice, starchy vegetables, fats and sugars, and moderate amounts of fish and fruit. During these periods her diet rarely contained eggs, milk, or green vegetables. By the end of each pregnancy she had gained only a few pounds in body weight. After the birth of each child, her appetite improved, she gained weight, and she felt fairly well until the next pregnancy.

The nausea, loss of appetite, and vomiting during this last pregnancy had been unusually severe. By the seventh month she was too weak to take care of her house and children and spent most of the time in bed. Severe burning and soreness of the tongue and diarrhea with frequent watery brown stools developed about six weeks before the birth of the child. Her husband had noticed that she was extremely pale.

A neighbor delivered the infant. Since delivery, the mother had not regained her appetite or her strength. She had not been able to do her housework or to take care of her children. For several days prior to her admission, she had been in bed and had seemed to be "out of her head." She had refused all foods except a small amount of milk and soup.

The infant weighed 5 lb. (2,268 gm.) at birth. Although the mother's supply of breast milk was scanty, feedings were given at frequent and irregular intervals during the day and night. The father said the infant had not been well since birth. She looked "like wax," cried when awake, and looked "like dead" when asleep.

Physical examination of the mother showed a pale, emaciated woman who appeared to be critically ill. She was too weak to sit up in bed. Her general pallor was striking. The skin, in addition to being pale, had a slightly yellowish tint. Her tongue was fiery red and smooth. The conjunctivas were pale and the scleras muddy.

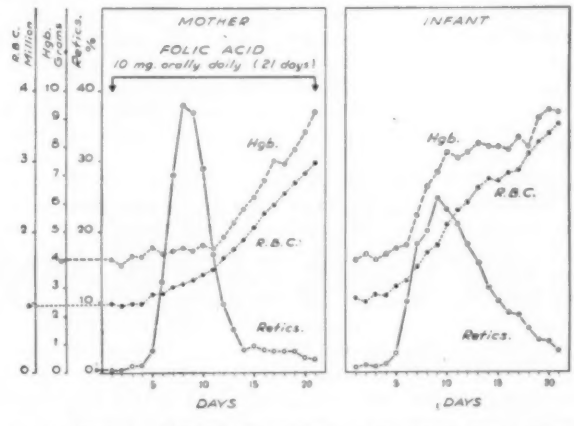


Fig. 1 (case 1).—Hemopoietic response of a mother with macrocytic anemia and her nursing infant with megaloblastic anemia, following administration of folic acid to the mother.

The infant was emaciated, pale, and weak. The buccal mucosa and the tip and border of the tongue were reddened. She was listless and appeared almost lifeless. Frequently she awakened, cried, and aroused the mother, who then tried to nurse her. Bowel movements were frequent and the stools liquid and brown.

Repeated gastric analyses showed free hydrochloric acid in the gastric juice of the mother. An oral glucose tolerance test showed a normal curve. The stools were watery and brown; they contained no ova, parasites, or blood. The peripheral blood findings on admission were as follows: red blood cells 990,000 and hemoglobin 4.0 gm. (26%), with 2% reticulocytes. The bone marrow showed characteristic megaloblastic arrest. The stools of the infant were watery and brown; they contained no ova, parasites, or blood. Her peripheral blood values were as follows: red blood cells 1,050,000 and hemoglobin 4.0 gm. (26%), with 0.6% reticulocytes. The respective diagnoses were macrocytic anemia of pregnancy and megaloblastic anemia of infancy.

After the base line studies were completed, the mother was given 10 mg. of folic acid orally daily for 21 days. As can be seen in figure 1, there was an excellent hemopoietic response.

1. Zuelzer, W. W.: Folic Acid Therapy in Anemias of Infancy and Childhood, J. A. M. A. **131**: 7 (May 4) 1946.

2. Angulo, J. J., and Spies, T. D.: Determination of Folic Acid Content of Food Usually Consumed by Patients with Tropical Sprue, Am. J. Trop. Med. **27**: 317, 1947.

3. Spies, T. D.: Observations on Macrocytic Anemia Associated with Pregnancy, Surg., Gynec. & Obst. **89**: 76, 1949.

The reticulocytes reached a peak of 37.9% on the eighth day of therapy, and thereafter the red blood cell count and hemoglobin increased rapidly. When she was discharged, on the 21st day of therapy, her blood values were as follows: red blood cells 2,990,000 and hemoglobin 9.2 gm. (60%), with 1.8% reticulocytes. Although given no therapy, the nursing infant likewise had a striking hemopoietic response. The reticulocytes increased to 24.6% eight days after start of folic acid therapy for the mother. By the time the mother had had folic acid for 21 days the infant's blood values were as follows: red blood cells 3,510,000 and hemoglobin 9.2 gm. (60%), with 2.8% reticulocytes.

The clinical response in both the mother and in the infant was dramatic. Three days after therapy was initiated the mother sat up in bed, ate all the food on her trays, and began talking to other patients in the ward, whereas prior to therapy she lay quietly in bed, seemed disinterested in her surroundings, and refused all food except a little soup. The soreness of her tongue began to decrease on the third day of therapy, and from this time on her improvement was rapid. All soreness of the tongue disappeared by the fifth day, and she began eating tremendous amounts of food. Within a few days her supply of breast milk was abundant. The diarrhea began to subside on the sixth day of therapy, and by the eighth day the stools were normal. The fourth day of administration of folic acid to the mother, a definite improvement in the infant's condition was obvious. The redness of the buccal mucosa had faded, her skin was slightly pink, and she was less fretful. Her appetite increased, and, when the mother's supply of breast milk increased, the infant was put on a normal feeding schedule. The diarrhea disappeared after the mother had received folic acid for seven days. By the time the mother had been treated for three weeks, the infant had gained 23 oz. (652 gm.); her color was good, and, although small, she appeared to be normal and healthy.

When the mother and child were discharged from the hospital, the mother was instructed as to what foods she should eat and how the child should be fed. They returned at weekly intervals for follow-up studies. Six months after they were discharged the mother's peripheral blood values were as follows: red blood cells 4,260,000 and hemoglobin 13.2 gm. (85%), with 0.2% reticulocytes. The infant's blood values were as follows: red blood cells 4,100,000 and hemoglobin 11.8 gm., with 0.8% reticulocytes. The mother had gained 22 lb. (10 kg.), and both she and the child appeared to be in excellent health.

The family moved to another town, and we did not see the mother or child again for 12 months. At this time the child still appeared to be in perfect health, but the mother complained of weakness, diarrhea, and soreness of the mouth and tongue. She was in the fourth month of her sixth pregnancy. During the first month she said she had lost her appetite, and

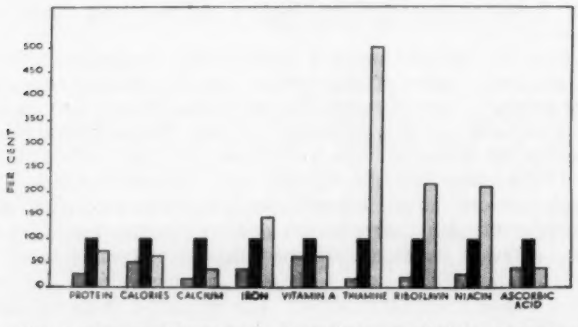


Fig. 2 (case 1).—Comparison of nutrients supplied by diet of patient with macrocytic anemia of pregnancy with (dotted areas) and without (areas with diagonal lines) dried yeast and recommended allowances (black areas). (Allowances recommended by the Council on Foods and Nutrition, National Research Council. The level for folic acid has not yet been determined.) The patient's diet was almost devoid of folic acid by analysis.

watery diarrhea, nausea, and occasional vomiting developed and persisted. She lost strength rapidly and had lost 12 lb. (5.4 kg.). Her peripheral blood values were as follows: red blood cells 2,100,000 and hemoglobin 6.9 gm. (44%), with 1.0% reticulocytes. She was admitted to the hospital and given 3 oz. (85 gm.) of dried yeast per day. Figure 2 shows the effect of the yeast supplement on the nutritive value of her diet. A prompt hemopoietic response followed this treatment. The reticulocytes reached a peak of 16% on the eighth day of therapy. Subsequently the red blood cells and hemoglobin increased, and by the 21st day of therapy the peripheral blood values were as follows: red blood cells 3,410,000 and hemoglobin 10.6 gm. (69%), with 1.8% reticulocytes. The glossitis, diarrhea, nausea,

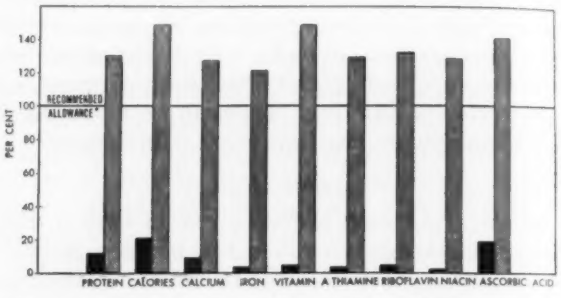


Fig. 3 (case 2).—Intake of nutrients before (black areas) and after (areas with diagonal lines) corticotropin therapy of leukemia in a 3½-year-old child. (*Recommended by the Council on Foods and Nutrition, National Research Council.)

and vomiting promptly disappeared. Her appetite increased, and by the time she was discharged, after three weeks of treatment, she had gained 15 lb. (6.8 kg.).

When she was discharged, a maintenance dose of 1 oz. (28 gm.) of dried yeast daily was prescribed. Since she lived too far away to make frequent trips in for follow-up studies, she was asked to write us in regard to her condition. Twelve months later she wrote to say that she had given birth to a 9 lb. (4,082 gm.) infant and that both she and the child were well. Ten months later she returned for blood studies and brought the two youngest children. She and both children appeared to be in excellent health. The blood values of both children were normal for their ages, and mother's blood values were as follows: red blood cells 4,370,000 and hemoglobin 13.6 gm. (88%), with 1.0% reticulocytes.

CORTISONE AND CORTICOTROPIN (ACTH)

The effects of cortisone and corticotropin are so broad that no satisfactory understanding of their action has yet been described. It would be presumptuous to assume that we could, in this comparatively brief paper, give a necessary comprehensive clinical review or even stress the most intriguing theories involving their physiological or biochemical action. Because we have worked for many years in the field of nutrition and because these substances have physiological effects associated with the science of nutrition, it seems fitting that we point out how the food intake is greatly augmented after the administration of these substances to a child with an acute disease. Many tissues of the body exhibit great sensitivity to these hormones. Exogenous cortisone and corticotropin have already been shown to have therapeutic application in diseases arising in many tissues. The present communication is concerned solely with those diseases affecting children and is based on our experience in treating children with rheumatoid arthritis, rheumatic fever, psoriasis, lupus erythematosus, various allergies, pemphigus, inflammatory eye disease, acute leukemia, and adrenal cortical hypofunction. Corticotropin is a specific stimulant for the adrenal cortex and initiates the formation of a number of corticoids by the glands. Physiological agents such as cortisone and compound F are vitally concerned with the mobilization and utilization of energy and with the well-being of the cells of the body, with the result

that they prevent or reverse the adverse reactions produced by a wide variety of diseases. It is not the purpose of this communication to report the specific effects of these hormones on the symptoms as such. We are concerned with the vital change in the patient that causes him to improve clinically and to show a tremendous interest in food. The example chosen is a patient with leukemia. Not all patients with acute leukemia show initial, or for that matter any, benefit.[4] In all our cases the treatment has failed to save life. When treatment has been interrupted, a relapse has occurred; in our experience, such treatment has never produced a full remission. Repeated examinations of the peripheral blood and of the bone marrow always show that the disease still exists. This is illustrated in case 2.

CASE 2.—A. F., a 3½-year-old child, had been well until three months prior to his admission, when a sore throat, swollen glands in the neck, and temperature of 38 to 39 C (100.4 to 102.2 F) developed. He was given various antibiotics, but the sore throat was not relieved, the glands in his neck remained swollen, and his temperature did not return to normal. Soon after the onset of the illness, he lost his appetite, lost weight, and began having severe pains in his legs. A month prior to his admission, purpuric spots appeared on his elbows, knees, and thighs.

Physical examination on admission showed an extremely pale child who appeared weak and very ill. He rarely opened his eyes or responded when spoken to and moaned and cried almost constantly. The glands in his neck and groin were enlarged. There was no enlargement of the spleen. The liver was one fingerbreadth below the costal margin.

The peripheral blood values were as follows: red blood cells 2,990,000, white blood cells 12,300, hemoglobin 7.5 gm. (48%), and no reticulocytes. The differential count showed 4 polymorphonuclear leukocytes, 1 myelocyte, 1 staff cell, 1 basophil, 2 eosinophils, and 91 lymphocytes. The erythrocyte sedimentation rate was 44 mm. per hour.

After the base line studies were completed, corticotropin, in doses varying in size from 10 to 20 mg., was administered intramuscularly four times daily. A sudden and tremendous increase

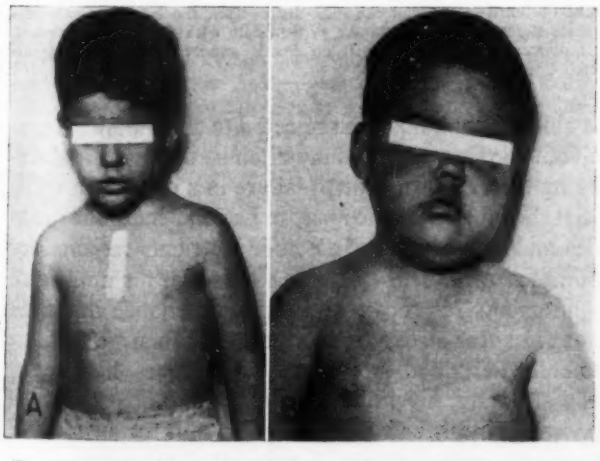


Fig. 4 (case 2).—Before (*A*) and after (*B*) the appearance of "moon face" in a 3½-year-old boy treated with corticotropin.

in appetite occurred on the third day of corticotropin therapy. The patient ate all the food offered him at mealtime, asked for additional servings, and requested foods at frequent intervals between meals, whereas he had been taking only small amounts of ice cream, milk, and fruit juice (fig. 3). On the fourth day he sat up in bed, began to take an interest in his surroundings, and played a little. Two days later the pain in his legs was relieved; he got out of bed and began playing around the ward. After a week of corticotropin therapy, the pain and inflammation of the joints disappeared, and the purpuric spots began to fade; two days later they had disappeared. By this time his temperature was normal, the glands in his neck and groin had decreased 50% in size, and he appeared and behaved like a normal, healthy child. In the period of 10 weeks he was given a total of 2,370 mg. of corticotropin; during this time he continued to have a tremendous appetite and a great increase in body weight. Since he exhibited the characteristic "moon face" (fig. 4) that has been attributed to excess intake of corticotropin,

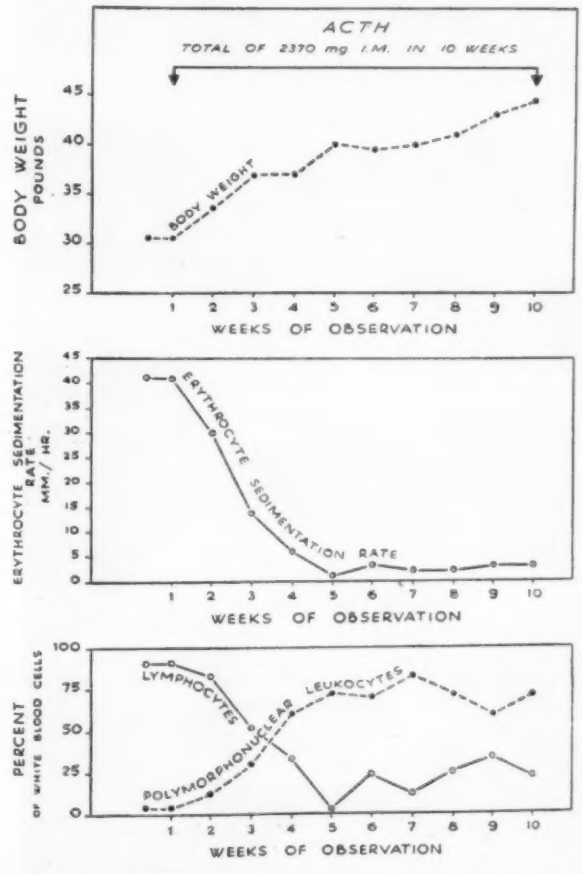


Fig. 5 (case 2).—Effect of corticotropin therapy on certain blood values and on body weight in a child with acute leukemia.

he was given no treatment of any kind for one week. Corticotropin was then administered in a dosage of 30 mg. daily. After a week treatment was discontinued. Clinically the patient remained well for five weeks; then he began to relapse. He lost his appetite and vomited frequently, and petechiae appeared over his whole body. He began having pain in his legs and temperature of 38 to 39.5 C (100.4 to 103.1 F), and the glands in his neck and groin began to increase in size. He was given a total of 1,125 mg. of cortisone acetate intramuscularly over a period of 12 days, but there was no change in his condition. He then was given 40 mg. of corticotropin intramuscularly daily for 10 days. After two days there was moderate clinical improvement. His appetite increased, and he appeared more alert. This improvement lasted only two days; then his condition quickly grew worse. Bleeding from the gums began, and severe tachycardia developed; he died four days later.

4. Pearson, O. H., and others: ACTH- and Cortisone-Induced Regression of Lymphoid Tumors in Man: A Preliminary Report, Cancer **2**: 943, 1949. Farber, S., and others: The Effect of ACTH in Acute Leukemia in Childhood, Proceedings of the First Clinical ACTH Conference, Philadelphia, Blakiston, 1950, p. 328. Dameshek, W.; Saunders, R. H., Jr., and Zannos, L.: The Use of ACTH in the Treatment of Acute and Subacute Leukemia, Bull. New England M. Center **12**: 11, 1950. Stickney, J. M.; Heck, F. J., and Watkins, C. H.: Cortisone and ACTH in Management of Leukemia and Lymphoblastoma, Proc. Staff Meet., Mayo Clin. **25**: 488, 1950. Spies, T. D., and others: Response to Adrenocorticotropic Hormone and Cortisone in Persons with Carcinoma, Leukemia and Lymphosarcoma, Lancet **2**: 241, 1950. Suzman, M. M.; Goldberg, B., and Hirsch, H.: Cortisone in Acute Lymphoblastic Leukemia, Lancet **1**: 760, 1951.

This case shows that, in certain selected patients with acute lymphoblastic leukemia, the administration of corticotropin or cortisone acetate is followed by general improvement characterized by a great increase in the amount of food ingested, gain in body weight, and decrease in erythrocyte sedimentation rate and in hemorrhagic manifestations of leukemia. In the peripheral blood, the red blood cells, hemoglobin, and polymorphonuclear leukocytes increase and the lymphocytes decrease (fig. 5). A large number of megaloblasts appear in the bone marrow. At no time, however, is the bone marrow or the peripheral blood devoid of leukemic cells.

Basic Diet From 50th to 100th Day
A
Basic Diet Plus B Vitamins 50th to 100th Day
B
Basic Diet Plus Yeast 50th to 100th Day
C
D
BASAL DIET PLUS — CASEIN, MINERALS
E
BASAL DIET PLUS — CASEIN, MINERALS, VITAMINS

Fig. 6 (case 3).—All the rats were given the stock diet for 50 days; then they were given the test diet plus various supplements for the next 50 days, with the results shown above.

GROWTH PROMOTING FACTORS

It is obvious that many dietary elements have specific functions relating to the growth of a child and to the maintenance of his health and well-being. For 15 years we have been studying the effects of chronic malnutrition on adults and children attending our clinic. In varying degrees the children are underweight and underdeveloped for their ages. After the administration of proper vitamins, such as riboflavin, nicotinamide, and thiamine hydrochloride, their appetites and weights usually increase. It must be stressed that this increase in appetite and weight gradually levels off unless the diets are improved. To improve these diets, dried yeast, casein, or dry milk solids are often added.

In many instances, however, the superficial and more obvious manifestations of growth failure induced by chronic malnutrition may be obscured by the intensity of the inherent growth-promoting factors operating within the child. Case 3 has been selected to show how the general outward appearance may often be deceiving in determining the extent of growth failure in a child consuming a diet shown by biologic assay to be deficient in the quantities and proportions of essential nutrients required for optimal growth. This case also shows that the nutritional requirements for repair and for maintenance are often not as exacting as they are for growth. When we first saw the child described in the following report, his general body structure, height, and weight were not particularly abnormal for his age. As we studied him for a period of 80 months, many interesting features of his nutritive and growth failure made their appearance as can be seen from his case history.

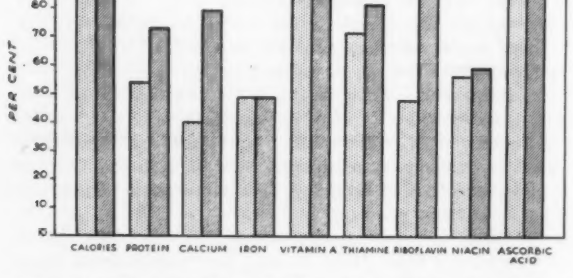


Fig. 7 (case 3).—Effect of supplement of nonfat, dry milk solids on nutritive value of diet of E. T. The dotted areas indicate the average daily intake of nutrients without the milk supplement; the cross hatched areas indicate the average daily intake of nutrients with the milk supplement. (*Recommended by the Council on Foods and Nutrition, National Research Council.)

CASE 3.—E. T., a 12-year-old white boy, was first seen by us when he was 5 years of age. He is the fourth child in a family of 10 children ranging in age from 1 to 16 years.

He weighed 11¾ lb. (5,330 gm.) at birth, cut his first tooth at 7 months of age, sat alone at 10 months, and walked and talked at 12 months. He was breast-fed until he was 15 months old. He was then given the same foods as the rest of his family. The diet consisted chiefly of cornbread, biscuit, fat pork, sugar, and, occasionally, turnip greens, corn, tomatoes, and berries in season.

Rarely did he receive any milk, eggs, meat, fish, or cheese. To determine the growth-promoting capacity of this diet it was scaled down to the rat level and fed to a group of young animals. We could not ascertain precisely the small amounts of berries and vegetables that the child occasionally ate, so we did not incorporate such materials into the diets of the rats. The basic foods in the diet of the rats were present in the same relative proportion as in the diet of the child. We prepared the diet for the rats by adding a small amount of water, heating the mixture slowly until it was dry, and then grinding it into small pieces in a food chopper.

Sixty male rats, 50 days of age, were divided arbitrarily into five groups of 12 each. For 50 days each rat was given the basic diet eaten by the child. During this period, 12 rats received only the basic diet and water; 12 received the basic diet and water plus daily supplements of thiamine hydrochloride, nicotinic acid, riboflavin, pyridoxine, pantothenic acid, choline, and inositol; 12 received the basic diet and water plus 1.5 gm. of dried yeast daily; 12 received the basic diet and water plus casein and a mineral mixture daily; and 12 received the basic diet and water plus daily supplements of casein, a mineral mixture, and the synthetic vitamins mentioned above. Figure 6 shows a representative rat from each group. As can be seen, the rats receiving only the basic diet and water were small in size and emaciated in appearance and had soft and flabby muscles. The rats receiving the supplement of synthetic vitamins grew to a larger size and appeared healthier than those on the basic diet alone. The rats receiving the dried brewers' yeast powder appeared considerably healthier than either the group receiving the basic diet alone or the group receiving the basic diet plus the synthetic vitamins. The rats receiving casein and minerals appeared larger and stronger than those receiving the basic diet alone, but only those rats receiving the casein, minerals, and vitamins appeared to be normal in size and health. Certainly one is justified in concluding that the diet eaten by this child is inadequate for the proper growth and nutrition of young rats and that synthetic vitamins alone were not as effective in promoting growth as was the addition of dried yeast or a supplement of casein, minerals and vitamins.

Physical examination of the child when he was 5 years of age showed him to be fairly well developed. He had bilateral cheilosis, glossitis of pellagra, and marked injection of the conjunctivas and scleras. His mother said that he had had "cracks" at the corners of his mouth most of his life and that frequently his tongue was sore and red.

During the following three years, active bilateral cheilosis and glossitis were present much of the time. His mother complained that he was "fractious." His teacher stated that he did not concentrate, that his grades were poor, and that he was "quarrelsome." After a period of observation of three years and nine months, he was given a dietary supplement of dry, nonfat milk solids equivalent to 3 qt. (3 liters) of skimmed milk a week by methods already described.[5] The effect of this supplement on the nutritive value of his diet is shown in figure 7.

The growth progress of this child as measured by the Wetzel grid technique is shown in figure 8. At 5 years of age he had attained developmental level 25, had a physique rating of "stocky," and was proceeding on a fast auxodrome. His developmental age (Wetzel) was 5 years 10 months. In the three years and nine months of observation he progressed at an over-all rate of approximately one developmental level per month. His physique rating changed from "stocky" to "good." There was no corresponding persistent auxodromal lag, thus indicating that he was increasing in stature at a proportionately faster rate than he was increasing in weight. His grid curves were characterized by recurrent periods of retarded growth followed by periods of recovery. These followed a definite seasonal pattern; growth retardation being observed each summer, recovery each fall and winter. Similar manifestations were observed in the growth curves of each of his six brothers and two sisters, who received the same general diet and each of whom has had cheilosis. On several occasions the brothers and sisters have been given either synthetic riboflavin or dried yeast, as a result of which their symptoms were ameliorated. After the administration of these materials was discontinued, the cheilosis gradually reappeared. In each child, the lesions were worse in the summer than at any other season of the year.

The milk supplement was started when the child was 8 years 9 months of age. In the 35 months since he has been receiving the supplement, he has maintained essentially the same general grid pattern established during the presupplementation period. During the first year in which he was given the milk supplement, little change was noted in his cheilosis, glossitis, disposition, or school grades. In the past year there has been a gradual improvement in the lip and tongue symptoms. His mother says that his disposition has improved greatly. His teachers say that he can concentrate better on his studies, that his school grades have improved, and that his behavior is excellent.

It is apparent that the comparatively small amount of milk given as a dietary supplement failed to increase the growth rate of this child over and above that noted during the presupplementation period. The addition of the dietary supplement served, however, to maintain the fairly rapid rate of growth characteristic of this child in the face of increasing requirements corresponding with an increase in age. Analysis of the nutrient value of his basic diet and the failure of this diet to support the growth of young rats suggests that the growth rate of this child, while admittedly rapid, was still below optimal. The gradual diminution of severity and duration of the anatomic lesions indicative of the nutritional deficiency

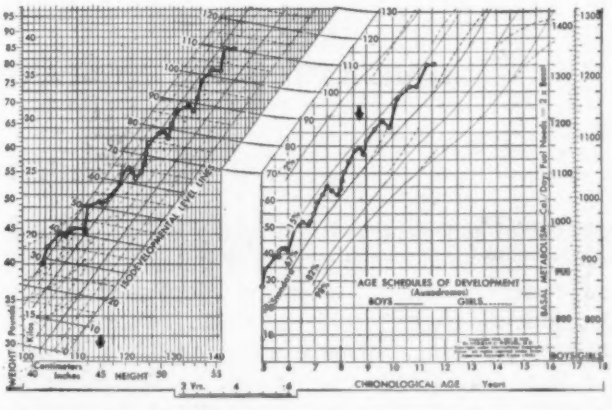


Fig. 8 (case 3).—Growth progress of E. T. as measured by the Wetzel grid technique. The arrow indicates the point at which the milk study began.

state suggests that the nutritional requirements for repair and maintenance in this child were not as exacting as those for optimal growth.

COMMENT

The delicate interrelationship of the various nutritional, hormonal, and hereditary factors is gradually unfolding. From the beginning, it has been obvious that hereditary factors, various diseases, nutrition, and many other conditions determine the course of physical growth and development. Certainly the relative importance of each has not been determined and perhaps varies from child to child and from time to time. The effect on the infant of improper nutrition in the mother is shown in case 1. In this case it is shown that folic acid is a nutrient having a specific function in the well-being and health of the infant. Case 2 shows in a most dramatic way how

5. Mann, A. W.; Dreizen, S.; Pyle, S. I., and Spies, T. D.: The Red Graph and the Wetzel Grid as Methods of Determining the Symmetry of Status and Progress During Growth, J. Pediat. **32**: 137, 1948. Spies, T. D., and Dreizen, S.: Effect of Milk Supplements on Growth of Children with Nutritive Failure, ibid. **34**: 393, 1949. Dreizen, S.; Currie, C.; Gilley, E. J., and Spies, T. D.: The Effect of Milk Supplements on Growth of Children with Nutritive Failure: Height and Weight Changes, Growth **14**: 189, 1950.

the administration of corticotropin (ACTH) to a child with a lethal condition, namely, leukemia, is followed by a tremendous increase in appetite, with result that the child eats voraciously and rapidly increases his body weight. Case 3 is an example of many children we have studied with an erratic growth pattern whose diets have been high in carbohydrates and fats and low in proteins, minerals, and vitamins. This case illustrates the principle that the effects of prolonged nutritive failure on the growth progress of a child may often be subtle and devious and marked by a high degree of individual variation; detection of these effects may require extensive study.

The seasonal occurrence of episodes of retarded growth in children with chronic nutritive failure corresponds to the same periods of the year when the children and their parents have clinical manifestations of nutritional deficiency diseases. In a series of 41 children, we found that the addition of a weekly supplement of 6 qt. (6 liters) of milk (twice the amount given the child in case 3) for 20 months produced an increase in the rate of growth in 73%, an increase in the speed of development in 54%, an improvement in the direction of development in 34%, and an improvement in the quality of growth in 27%. We have also demonstrated that the children receiving the milk supplements gained an average of 1.23 cm. in height and 1.35 kg. in weight over and above the gains made by the children not receiving the supplements during the 20-month period. These values were equal to 11.8% and 22.1% of the respective mean height and weight gains made by the children receiving the added milk during the supplementation phase of the study. These findings illustrate that the retarding effects of prolonged nutritive failure on the growth progress of children are, to a large extent, reversible, provided that the dietary therapy is ample enough to overcome the accumulated deficiencies of many years' standing.

Such observations as those reported here, along with the studies being done in many other places, will eventually bring much valuable information. The young science of nutrition will then be able to recommend more comprehensive principles, which, when judiciously applied, will produce even more gratifying results in this field.

620 S. 20th St. (Dr. Spies)

LIMITATIONS OF SYMPATHECTOMY IN TREATMENT OF DIASTOLIC HYPERTENSION

Geza de Takats, M.D., Chicago

Essential hypertension is a progressive disease that is usually discovered by the general practitioner or at preemployment, insurance, or preinduction examinations. Obviously the majority of patients with hypertension do not need operation, but early intensive study is imperative, since a decision to operate may well depend on the discovery of progressive vascular disease in the cerebral, retinal, cardiac, renal, and peripheral vascular areas.

The purpose of this paper is to present some tentative conclusions about when to operate, what type of operation to do, and how to interpret results in patients who have diastolic hypertension. Excluded from this discussion are patients with well-defined causes of hypertension, such as coarctation of the aorta, unilateral renal disease, pheochromocytoma, corticoadrenal tumors, and the full-blown Cushing's syndrome. This leaves a large number of patients affected by a hypertensive cardiorenal disease, for which a variety of etiologic factors, classifications, and methods of management have been proposed. Up-to-date information on trends of experimental investigation and clinical studies may be found in the transactions of recent symposiums.[1]

Since 1943 562 patients have been operated on by our group in three institutions.[2] Reports on these operations have been published from time to time [3] and will be referred to only when they apply to the problem at hand. Not only does the patient material vary from institution to institution or even within the same institution from year to year but so does the classification of patients, their study, and the evaluation of results. Expertly analyzed statistics may help to minimize some of the variables but they often obscure the individual prognoses. For this reason from the patients operated on during 17 years, a group of 55 who were operated on in 1946 with varying indications, technique, and five year follow-up are considered here. The pilot study has confirmed the originally expressed conservatism in advising operation only in rigidly defined cases and rejecting patients in whom operation has seemed either unnecessary or futile.

OBJECT OF PREOPERATIVE STUDY

Elsewhere [4] the work-up of hypertensive patients has been outlined, and only the summary sheet is presented here (fig. 1). The object of such a study is to estimate the emotional and the regional vascular status of the hypertensive person and to evaluate the importance of neurogenic, endocrine, and renal factors in each case. Such a viewpoint is based on the assumption that human

Read at the Fifth Clinical Session of the American Medical Association, Los Angeles, Dec. 4, 1951.

From the Department of Surgery, University of Illinois College of Medicine, and St. Luke's Hospital.

The cooperation of Drs. George W. Scupham, Richard C. Gamble, and James C. Clark, and Mrs. Loraine Ade Hanlon made this long-range study possible.

1. (*a*) Bell, E. T., editor: Hypertension, Minneapolis, University of Minnesota Press, 1951. (*b*) Wolff, H. G.: Life Stress and Bodily Disease—Formulation, A. Res. Nerv. & Ment. Dis. Proc. **29**: 1059, 1950. (*c*) Conference on Factors Regulating Blood Pressure, Transactions of the 1st to 3rd Conferences, New York, Josiah Macy, Jr., Foundation, 1947 to 1949.

2. St. Luke's Hospital and the Research and Educational Hospitals, University of Illinois, Chicago, and the Veteran's Administration Hospital, Hines, Ill.

3. (*a*) de Takats, G.; Heyer, H. E., and Keeton, R. W.: Surgical Approach to Hypertension, J. A. M. A. **118**: 501 (Feb. 14) 1942. (*b*) de Takats, G.; Graupner, G. W.; Fowler, E. F., and Jensik, R. J.: Surgical Approach to Hypertension: Second Report, Arch. Surg. **53**: 111 (Aug.) 1946. (*c*) de Takats, G., and Fowler, E. F.: Surgical Treatment of Hypertension: Neurogenic Versus Renal Hypertension from Standpoint of Operability, Surgery **21**: 773, 1947. (*d*) de Takats, G.; Julian, O. C., and Fowler, E. F.: Surgical Treatment of Hypertension, ibid. **24**: 469, 1948.

4. de Takats, G.: Selection of Hypertensive Patients for Sympathectomy, Chicago M. Soc. Bull. **53**: 797, 1951.

essential hypertension is probably not the equivalent of any of the experimental hypertensions produced through neurogenic, corticoadrenal, or renal mechanisms, but that these well-defined experimental procedures simply illustrate the mechanisms whereby arteriolar tone in the human can be raised. For example, an emotional stress originating from the cerebral cortex may operate through all three mechanisms and no doubt does, since hypothalamic stimulation will activate the sympathetics, mobilize corticoadrenal secretion through the mediation of the anterior pituitary, and demonstrably decrease renal blood flow.[1b] On the other hand, irreversible regional vascular damage in the kidney will produce all the known characteristics of experimental renal hypertension, but a neurogenic and adrenocortical factor may both be demonstrable. For this reason interpretation of the hypertensive study depends on clinical measurement of these three mechanisms.

The psychogenic factor is screened by an interview with a psychiatric social service worker, who in an hour or so takes the life history of the patient. As a result of such an interview the patient may have to be deferred from an operation even though he would otherwise be eminently suitable. After establishment of the proper physician-patient relationship,[5] the patient may become

HYPERTENSIVE WORK-UP SUMMARY SHEET—SURGERY IV

Name_____ Hospital #_____

Eyegrounds_____ Date_____ Age_____ Sex_____

ELECTROCARDIOGRAM_____ URINE ANALYSIS

2 meter chest film_____ Color_____ Diacetic_____ Sp. gr._____

I.V. Pyelogram_____ Acetone_____ pH_____ Alb. _____

Urea Clearance_____/100 cc. of blood Sugar_____ Casts_____ Cells_____

25 min. P.S.P._____ Bact._____

Cold Pressor Test — Water Tolerance: Amount — Spec. gravity

Basal_____ Ceiling_____ Conc. spec._____

Time till return _____ min. Spec. 1 _____

Exercise Tolerance Test Spec. 2 _____

Basal _____ Maximum _____ Spec. 3 _____

Ephedrine Spec. 4 _____

Basal_____ Minimum_____ Spec. 5 _____

Histamine Test Spec. 6 _____

Basal _____ Maximum _____ Spec. 7 _____

Rumpel-Leede Test Spec. 8 _____

Petechiae per area_____ Sodium Amytal

Blood Count Basal_____ Minimum_____

RBC_____ WBC_____ Insulin Tolerance

Hg._____ Hematocrit_____ Fasting_____ 15 min._____

Kahn_____ Sed. Rate_____ 30 min._____ 60 min._____

Blood Chemistry 90 min._____ 120 min._____

Bl. Sugar_____ Urea N._____ Epinephrine-Eosinopenia

N.P.N._____ Cholesterol_____ Before_____ After_____

B.M.R._____ Percentual Drop_____

INTERPRETATION AND RECOMMENDATIONS:

Fig. 1.—Summary sheet of hypertensive work-up.

a suitable candidate for operation. Clinical evaluation of this factor is just as important as the study of the eyegrounds or the concentration-dilution test. Ignoring this factor is a potent cause of failure of the surgical treatment.[6] It has not been the experience of our group that psychoanalytic treatment effectively and permanently lowers blood pressure, even in young persons with labile blood pressures and little if any organic vascular damage.[3c] However, by better adjustment to their environment patients treated psychoanalytically may become useful members of society and, should operation be otherwise indicated, this stress may be borne without further insult to their emotional state.

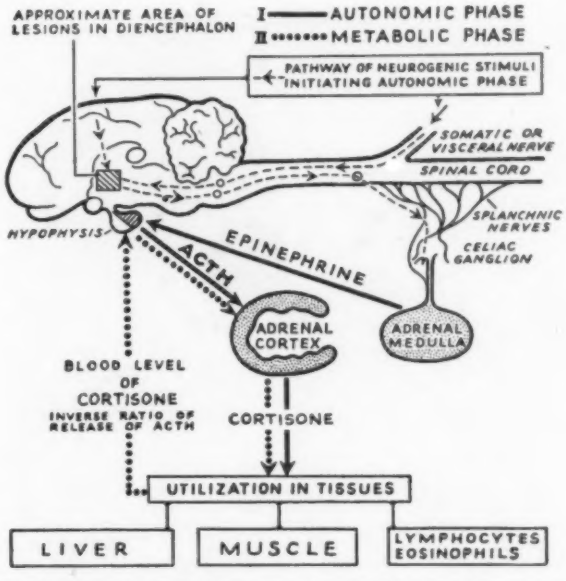


Fig. 2.—Proposed explanation of control of the secretion of adrenal cortical steroids. The diencephalon is activated by neurogenic stimuli from the cortex and by peripheral somatic or visceral afferent impulses which release epinephrine from the adrenal medulla. Epinephrine in turn mobilizes corticotropin (ACTH) from the anterior pituitary gland, which activates the adrenal cortex to secrete cortisone. This is the first autonomic phase. In the second metabolic phase corticotropin production is stimulated by the increased rate of utilization of cortisone. (McDermott, W. V.; Fry, E. G.; Brobeck, J. R., and Long, C. N. H.: Mechanism of Control of Adrenocorticotrophic hormone, Yale J. of Biol. & Med. **23**: 52, 1950.)

Regional Vascular Damage.—The grouping of patients with essential hypertension into four groups, based primarily on changes in eyegrounds but correlated with vascular damage elsewhere, is the contribution of Keith, Wagener, and Barker,[7] and no additional modification or subdivision has really improved it. While it is common clinical experience that the vascular damage in the eyegrounds does not parallel the vascular damage in other areas, notably in the brain and kidney and while transitional cases will always be encountered, this classification and particularly the correlated mortality and survival rates have served as a basis for comparison of operative results by which surgeons have showed that the operation favorably influences the survival rate.[8] Hypertensive encephalopathy, papilledema, cardiac decompensation, and severe angina with and without coronary thrombosis, to mention the advanced forms of regional vascular damage, have not been regarded as contraindications to sympathectomy. On the contrary, when done long

5. Ferris, F. B., and others: Life Situations, Emotions and Course of Patients with Arterial Hypertension, A. Res. Nerv. & Ment. Dis., Proc. **29**: 870, 1950.

6. de Takats, G.: Causes of Failure in Surgical Treatment of Hypertension, Angiology **1**: 547, 1950.

7. Keith, N. M.; Wagener, H. P., and Barker, N. W.: Some Different Types of Essential Hypertension: Their Course and Prognosis, Am. J. M. Sc. **197**: 332, 1939.

8. Peet, M. M., and Isberg, E. M.: Surgical Treatment of Essential Hypertension, J. A. M. A. **130**: 467 (Feb. 23) 1946. Smithwick, R. H.: Symposium on Recent Advances in Surgical Physiology: Surgical Physiology of Hypertension, S. Clin. North America **29**: 1699, 1949.

enough after the acute episode such decompensation has been regarded as an indication to prolong the patient's life and improve his vascular status. In essential hypertension when renal vascular damage is based predominantly on a fixed renal mechanism, the renal vascular condition has not been seen to respond to sympathectomy.

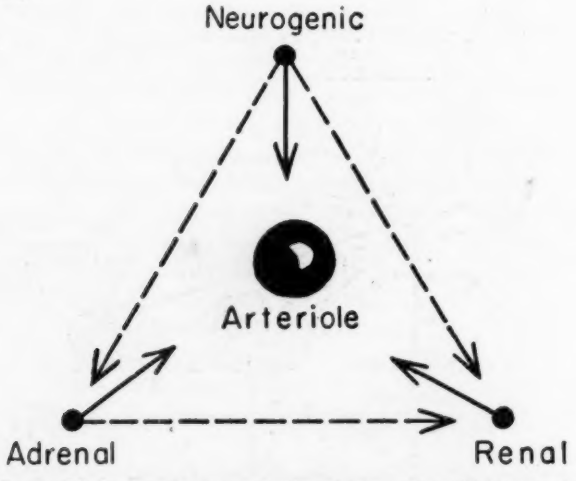


Fig. 3.—Mechanisms in the production of human essential hypertension. The neurogenic factor influences both other factors; the adrenal factor influences the renal one; whether the renal factor sensitizes the vessels to neurogenic hyper-reactivity is under debate. A combination of all these mechanisms is present in human essential hypertension.

The underlying vascular damage in hypertension is hyperplastic arteriolar sclerosis, which may be complicated later by atheromatosis. This arteriolar sclerosis is the result and not the cause of hypertension.[9] Pure arteriosclerotic hypertension, characterized by a rigid, atheromatous, and often calcified aorta, a high pulse pressure with a comparatively low diastolic pressure and a poor depressor mechanism in response to emotional, exertional and cold stimuli, has never been regarded as an indication for surgical intervention, although it is no doubt included in compilation of some mass statistics.

DETECTION OF THE THREE FACTORS THAT MAINTAIN HYPERTENSION

The Neurogenic Mechanism.—The single short painful cold stimulus of Hines [10] is the unsurpassed measure of vascular reactivity and, if determined in a basal state, reflects the vascular temperament of a person from birth to grave. As shown by the study of Reiser and Ferris,[11] even this short single stimulus may evoke a humoral response that cannot be abolished by tetraethyl ammonium chloride (TEAC). Since this substance acts at the ganglionic synapse, its effect on resting blood pressure is a better measure of neurogenic tone than the effect of barbiturates, which act on the hypothalamus. In the present series both drugs have been used, and a statistical correlation between them indicates good agreement.[12] Nevertheless, the dampening of central impulses from the hypothalamus inhibits activation of the hypothalamic-pituitary-corticoadrenal axis, and thus barbiturates may influence a non-neurogenic factor.

In 1947 Fowler and I [3c] objected to the term "neurogenic hypertension" on the basis that satisfactory sympathetic denervation in nonrenal hypertension need not lower the blood pressure appreciably. We suggested that hypothalamic-pituitary stimulation may not be decreased by splanchnicectomy. Increasing knowledge since that time indicates that our early stand in this matter was justifiable. A diagram of Long and co-workers (fig. 2) illustrates how a neurogenic stimulus may jump to the endocrine apparatus and may become a self-perpetuating mechanism. We suggested the term neuroendocrine hypertension in contradistinction to renal hypertension.

It is more likely that all mechanisms are simultaneously activated (fig. 3) by a central stimulus, including the renal than that hypertension is caused by the neurogenic mechanism alone. This is the reason the term "a neurogenic factor in hypertension" is preferable to "a neurogenic hypertension," which, as a continuous diastolic hypertension, in man cannot exist without involving the other two systems.

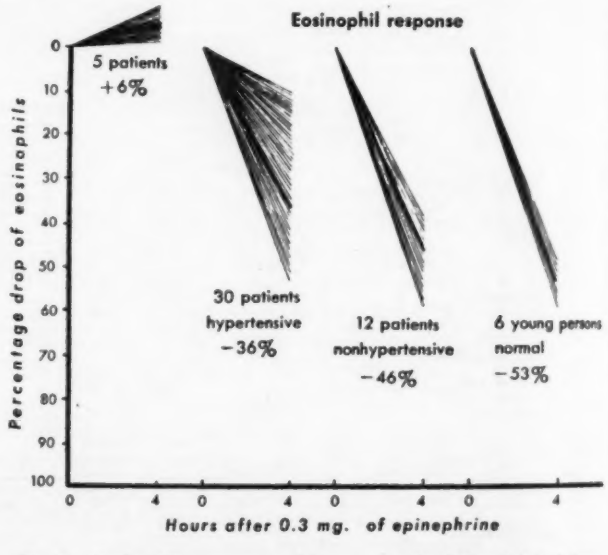


Fig. 4.—Eosinophil response to 0.3 mg. of epinephrine, injected intravenously in 200 cc. of isotonic sodium chloride solution. The first column shows patients with no response. They all had allergies. Thirty hypertensive patients (all before operation) showed less response than 12 normotensive patients. Six young normal persons showed the greatest response. (de Takats, G.: Causes of Failure in Surgical Treatment of Hypertension, Angiology 1: 457, 1950.)

Other methods for testing for vasomotor reactivity, such as the pressor response to carbon dioxide,[3b] to breath holding,[13] to posture,[14] and to exhaling with a

9. Moschcowitz, E.: Vascular Sclerosis with Special Reference to Arteriosclerosis: Pathology, Pathogenesis, Etiology, Diagnosis, Prognosis, Treatment, New York, Oxford University Press, 1942. Castleman, B., and Smithwick, R. H.: Relation of Vascular Disease to Hypertensive State: Adequacy of Renal Biopsy as Determined from Study of 500 Patients, New England J. Med. 239: 729, 1948.

10. Hines, E. A., Jr.: Significance of Vascular Hyperreaction as Measured by Cold-Pressor Test, Am. Heart J. 19: 408, 1940.

11. Reiser, M. F., and Ferris, E. B.: Nature of Cold Pressor Test and Its Significance in Relation to Neurogenic and Humoral Mechanisms in Hypertension, J. Clin. Investigation 27: 156, 1948.

12. Frew, J. L., and Rosenheim, M. L.: Labile Neurogenic Component of Hypertension: Comparison of Effects of Tetra-Ethyl-Ammonium Bromide and Rapidly Acting Barbiturate (Seconal), Clin. Sc. 7: 217, 1949.

13. Ayman, D., and Goldshine, A. D.: Breath-Holding Test: Simple Standard Stimulus of Blood Pressure, Arch. Int. Med. 63: 899 (May) 1939.

14. Smithwick, R. H., and Robertson, C. W.: Phenomenon of Hyperreactivity: Definition and Illustrations, Angiology 2: 143, 1951.

15. Wilkins, R. W.; Culbertson, J. W., and Smithwick, R. H.: Effects of Various Types of Sympathectomy on Vasopressor Responses in Hypertensive Patients, Surg., Gynec. & Obst. 87: 661, 1948.

16. Williams, A. H., and Schroeder, H. A.: Regional Vasomotor Tone in Normotensive and Hypertensive Dogs, Circulation 4: 706, 1951.

17. de Takats, G.: Corticoadrenal Factor in Hypertension, Surgery 26: 67, 1949.

closed glottis against measured resistance,[15] are useful. It should be emphasized, however that they simply measure vasomotor tone, which may be great in a patient with unilateral renal disease or chronic atrophic pyelonephritis. Thus a sympathectomy that has to extend to the third dorsal level [11] or a typical lumbodorsal sympathectomy [14] abolishes fluctuations of vasomotor tone and decreases vascular resistance in the mesenteric area, where it is the highest whether the hypertension is neurogenic or renal.[16]

The Corticoadrenal Mechanism.—This factor has been studied recently in hypertensive patients.[17] The insulin tolerance and the epinephrine eosinophilia [6] were used to estimate the participation of this mechanism. It would seem from the experience of Perera [18] that both sodium and the adrenal cortex are factors in blood pressure regulation, without the adrenals actually causing the hypertensive state. Efforts are now being made to depress the action of the adrenal cortex by sodium restriction, using potassium thiocyanate and sterols to counteract the sodium factor in the adrenal cortex. The surgeon can do a partial or total bilateral adrenalectomy with the aid

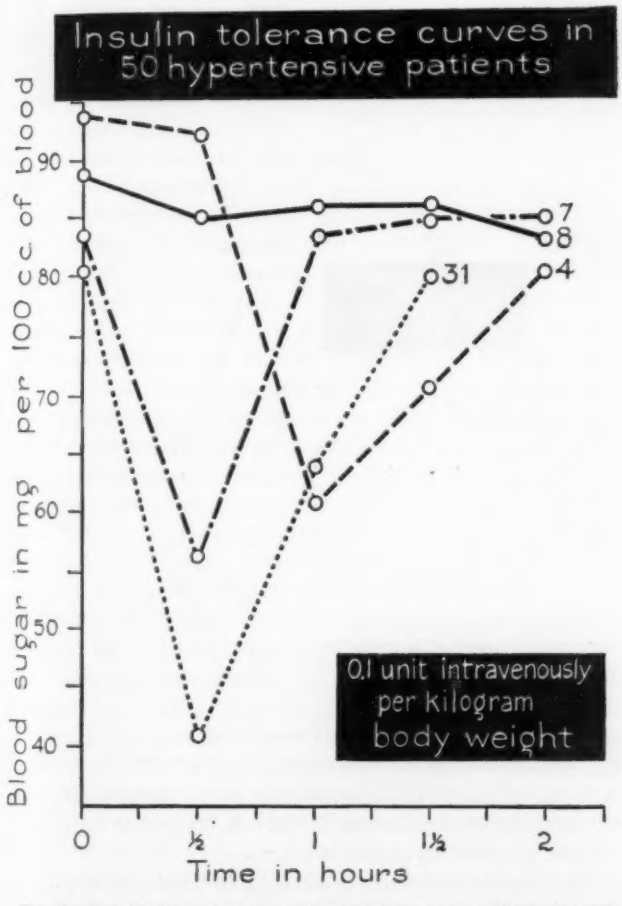


Fig. 5.—The insulin tolerance of hypertensive patients. Note that out of 50 patients, 8 showed no response and 4 a delayed response to insulin given intravenously. (de Takats, G.: Corticoadrenal Factor in Hypertension, Surgery **26**: 67, 1949.)

of replacement therapy,[18] but the indications are certainly not the same as those in Cushing's syndrome. In fact, our own data indicate that most patients with hypertension who come in for study at a late stage of their disease have a poor corticoadrenal reserve (fig. 4), perhaps in a phase preceded by increased activity.[19] Since regeneration of the adrenal remnant is dependent on anterior pituitary acceleration,[20] bilateral splanchnicectomy and dorsolumbar sympathectomy, combined with partial amputation of the adrenal glands bilaterally, seems indicated when corticoadrenal activity is demonstrable. The increased resistance to insulin, which is known to occur

Fig. 6.—Cortical adenoma removed with entire adrenal gland in a hypertensive man.

in Cushing's syndrome, in the diabetes caused by corticotropin (ACTH) in man, and in malignant hypertension has also been seen in a small group of patients with hypertension who are obese, who have a diabetic type of sugar tolerance curve, and whose symptoms fit Schroeder's description of a pseudo-Cushing's syndrome (fig. 5). While the adrenal glands are always examined during operation, such patients require specifically thorough examination and occasionally a cortical adenoma [17] (fig. 6) may be found.

The Renal Mechanism.—Hypertension resulting from bilateral primary disease of the kidney cannot be influenced by sympathectomy except by eliminating fluctuations of blood flow in a damaged kidney. The removal of one kidney affected by atrophic pyelonephritis or hypoplasia will have no effect if the other kidney has hypertensive damage. But in most patients it is the extent of bilateral nephrosclerosis that determines whether or not

18. Green, D. M.; Nelson, J. N.; Dodds, G. A., and Smalley, R. A.: Bilateral Adrenalectomy in Malignant Hypertension and Diabetes, J. A. M. A. **144**: 439 (Oct. 7) 1950. Zintel, H. A., and others: Subtotal Adrenalectomy in Treatment of Patients with Severe Essential Hypertension, Ann. Surg. **134**: 351, 1951.

19. Laragh, J. H., and Almy, T. P.: Changes in Circulating Eosinophils in Man Following Epinephrine, Insulin, and Surgical Operations, Proc. Soc. Exper. Biol. & Med. **69**: 499, 1948.

20. Ingle, D. J.: Control of Regeneration of Adrenal Cortex in Rat: Pituitary-Adrenal Function, Ass. for Advancement of Science, Washington, D. C., 1950.

hypertensive cardiovascular disease can be influenced by sympathectomy. This concept, recently supported by the work of Byrom and Dodson,[21] has been the basis of our consistent emphasis on the value of a water tolerance test (fig. 7) in determining operability, and on criteria that are far more rigid then if only the specific gravity range on concentration and dilution is utilized. Unless the patient shows one of the patterns of urinary output and specific gravity shown in the upper row of figure 6, the effect of sympathectomy, at least on the resting blood pressure, will be doubtful, since the renal mechanism will participate in the maintenance of diastolic hypertension and renal vascular damage is the least reversible of all types of vascular involvement.

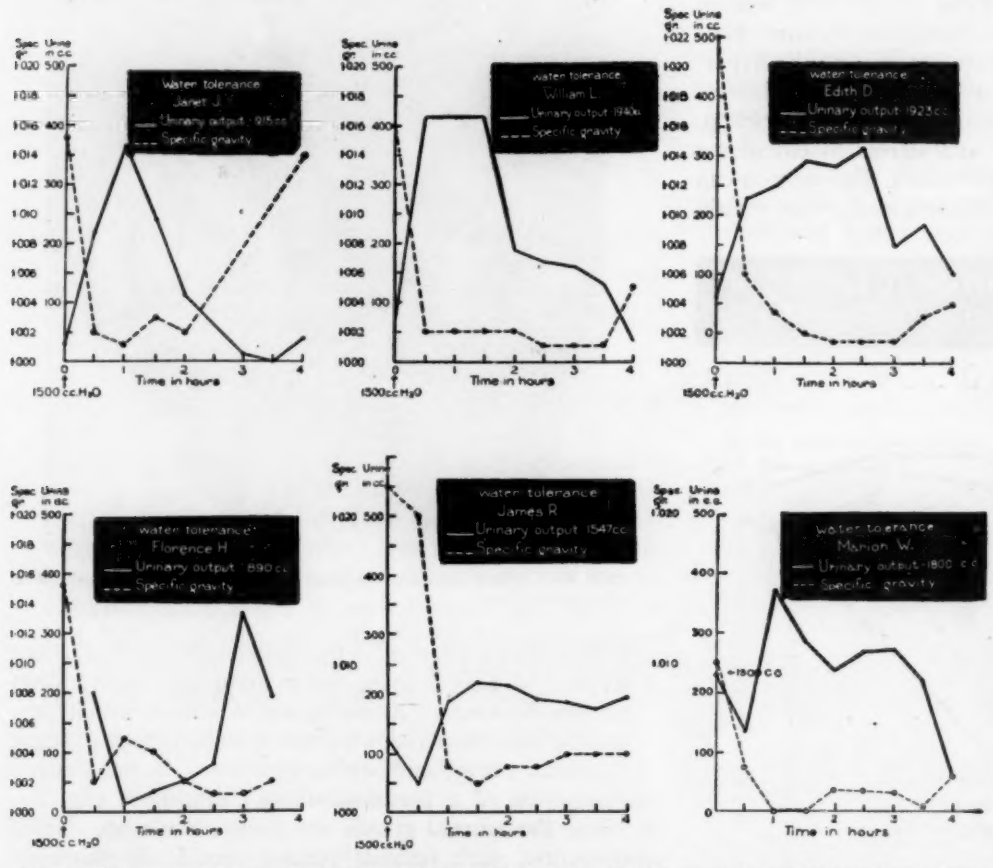


Fig. 7.—The water tolerance of hypertensive patients after ingestion of 1,500 cc. of water. The six patterns show a decreasing adaptability of the kidney to this stress. Correlation of the patterns with results obtained by dorsolumbar sympathectomy indicate that patterns 4, 5, and 6 constitute a contraindication to operation. (de Takats, G.: Cause of Failure in Surgical Treatment of Hypertension, Angiology 1: 457, 1950.)

Thus in a preoperative study of a hypertensive patient the emotional and regional vascular factors should be evaluated and the participation of the neurogenic, corticoadrenal, and renal mechanisms be weighed. The interrelation and the resulting vicious circle may be broken at several points, notably at the level of the sympathetics and in the adrenals. Revascularization of the ischemic kidney [22] has not resulted in improvement, at least in late stages of hypertensive cardiovascular renal disease.

Indication for Splanchnicectomy.—Consistently in the cases discussed three distinct indications for operation have been employed. An intermittent hypertension that can be reduced to normal by bed rest or by barbiturates may be watched expectantly, but, at the appearance of the earliest sign of vascular damage in the retina, heart, or kidney, sympathectomy should be undertaken for two reasons. In the first place, the patient has demonstrated his vascular vulnerability, which varies according to sex, heredity, environment, and possibly dietary factors. In the second place, operation in this stage is highly efficient in arresting the progress of vascular damage and in reducing the blood pressure to a normal level.

The second indication is the condition of a middle-aged hypertensive patient who shows diffuse vascular damage and whose moderately elevated diastolic pressure of 100 to 110 mm. Hg. shows a consistent rise in spite of medical management. Such a patient has irreversible arteriosclerosis and his diastolic pressure cannot be lowered below 100 to 110 mm. Hg. His prognosis will depend, as will be shown later, on the extent of renovascular damage and on the many uncontrollable factors that initiate vascular accidents in the brain and heart. Nevertheless, lowering of the diastolic pressure and the lessening of sudden rises of pressure that occur with exertion, undue emotion, cold, and posture change give such a patient better chance for survival than if the hypertension is not treated.[8]

The third indication is hypertension in the rapidly progressive premalignant phase or, should one believe in a malignant phase with papilledema, when the diastolic pressure is fixed but no advanced renal damage has occurred. In such patients the operation may be life-saving, but, as will be pointed out, the extent of the operation must be correlated with the severity of the disease so that in this stage more extensive procedures are in order.

The fourth indication is intractable headache resulting from hypertensive encephalopathy in patients with advanced renal damage. This palliative procedure is a limited one stage operation that is equivalent to a continuous venesection in diverting blood from the cerebral and retinal areas and aiding cerebral compensation. This fourth indication has been recognized very seldom as valid, but this situation does occur occasionally.

The extent of sympathetic denervation should be adapted to the patient's preoperative state (figure 8). The

21. Byrom, F. B., and Dodson, L. F.: Mechanism of Vicious Circle in Chronic Hypertension, Clin. Sc. 8: 1, 1949.

22. de Takats, G., and Scupham, G. W.: Revascularization of Ischemic Kidney, Arch. Surg. 41: 1394 (Dec.) 1940.

one stage supradiaphragmatic sympathectomy (Peet) has been used for palliation. The two stage subdiaphragmatic operation (Adson), with partial adrenalectomy, is now used for patients with marked corticoadrenal participation. Retropleural transdiaphragmatic extension is always possible. The two stage lumbodorsal sympathectomy and splanchnicectomy (Smithwick) is done in ju-

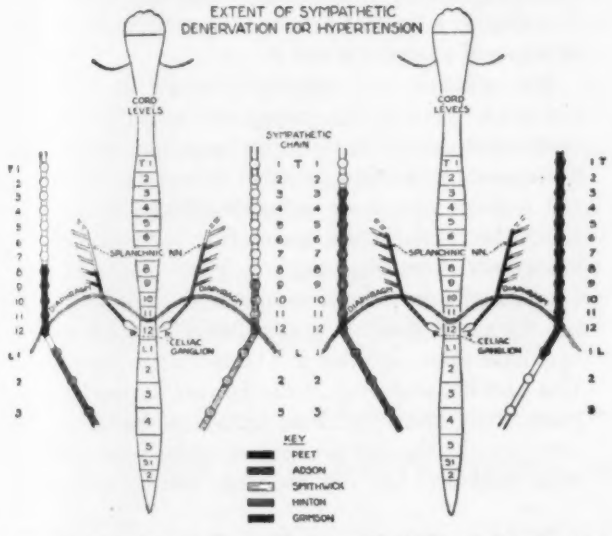


Fig. 8.—The extent of sympathetic denervation for hypertension as done by different surgeons. (de Takats, G.; Julian, O. C., and Fowler, E. F.: The Surgical Treatment of Essential Hypertension: IV. Case Selection and Technique as Influencing Result, Surgery **24**: 469, 1948.)

veniles with intermittent hypertension, in women with post-toxemic hypertension, and for progressive diastolic hypertension prior to the malignant phase; in other words, in the majority of patients. A transpleural, thoracic sympathectomy is done in patients with cerebral or cardiac decompensation, the lumbar chains being preserved to avoid postural hypotension. A two to four stage transpleural sympathectomy from the third dorsal to third lumbar nerve (Hinton)[23] is performed in the premalignant phase when there is adequate renal function. A total sympathectomy or sympathectomy with rhizotomy at the levels of D12, L1, and L2 [24] has not been performed in this series.

EVALUATION OF RESULTS

Three possible methods may be used for evaluation of results of sympathectomy in hypertension. Comparison of mortality and survival rates of patients treated by operation with the rates in the nonsurgical series of Keith, Wagener, and Barker [7] has been utilized by some clinics.[8] There are two objections to this method: first, the nonsurgical series may contain unusually advanced cases (fig. 9), and second, the survival rate per se does not represent the severe invalidism that patients suffer following a cerebrovascular accident or repeated coronary occlusion with status anginosis or bouts of decompensation. The second approach has been used by us in estimating the fall in blood pressure in three groups of patients treated surgically. The falls are averaged and compared to preoperative figures. Two valid criticisms of this method are that the preoperative blood pressures, no matter how often and how carefully taken, are invariably too high owing to preoperative stress, and, second, that such surveys assume that lowering of blood pressure is the only goal of the operation. Whether the blood pressure will be lowered is certainly the question asked by all patients prior to operation. Since the operation is intended for prevention or arrest of progress of cardiovascular renal disease, the height of the blood pressure does not indicate the improvement of eyegrounds, diminution of heart size, improvement in the cardiogram, or relief of increased vascular resistance in the mesenteric area. Therefore in evaluating the five year results to be presented here, I have simply separated those who have had a worth-while result from those in whom the operation has been considered to be not worth while. This introduces a purely subjective element in the judging of results, but it is the same subjective element that will lead the surgeon to advise sympathectomy in one case and not in another. It also enables the patient's family to have a preoperative prognosis that none of the statistical data dealing with trends, groups, means, or ambiguous results of analyses can supply.

The result was considered worth while in the first group with intermittent hypertension when the pressure fell to normal. The upper limit of normal diastolic pressure was considered to be 100 mm. Hg. on casual office examination, and the patient should have full working ability. In the second group of middle-aged patients with arteriosclerosis a worth-while result was postulated as stabilization of diastolic pressure at or below 110 mm. Hg., together with improvement or no deterioration of cardiovascular status and restoration of full working ability. Obviously, the patient may become disabled by an independent disease or an automobile accident that would not be attributed to the cardiovascular disease. In the premalignant group disappearance of papilledema, improvement of symptoms, and even being alive after five years with some earning power was counted as a worth-while result. Since all the palliative operations done in the malignant phase have been followed by death

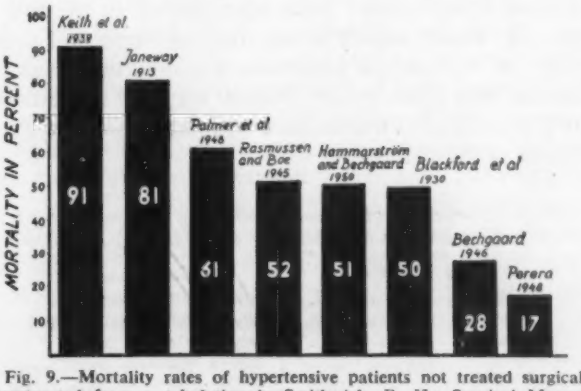


Fig. 9.—Mortality rates of hypertensive patients not treated surgically. Constructed from a tabulation in Smithwick, R. H.: Surgical Measures in Hypertension, Springfield, Ill., Charles C Thomas, Publisher, 1951.

within five years, the improvement in this group can only be judged by relief of intractable headache, palpitation, and angina. This group has not been evaluated because five years ago only three such patients were operated on. At present drugs, diets, venesection, or partial adrenalec-

23. Hinton's approach is retroplural, but the extent of the operation is the same.

24. Ray, B. S., and Console, A. D.: Evaluation of Total Sympathectomy, Ann. Surg. **130**: 652, 1949.

tomy may entirely eliminate the use of sympathectomy for malignant hypertension.

From the above description the group without worthwhile results can readily be defined. The results in the first and third groups after five years are very satisfactory (table 1). Dissatisfaction with the results in the second group led to examination of the causes of failure, and the importance of studying renal function became apparent. A table presented in a previous study indicates that there are some failures, of course, as a result of insufficient denervation or subsequent cardiovascular accidents, but the majority of them were unexplained (table 2). Correlation of these failures with the pattern of water tolerance [25] (table 3) shows the importance of an irreversible renal mechanism in vitiating the result of sympathectomy. This is the main limiting factor of sympathectomy.

TABLE 1.—*Results of Lumbodorsal Sympathectomy for Hypertension After Five Years*

Group	No. of Cases	Results: Worthwhile	Results: Not Worthwhile
1	15	12	3
2	31	21	10
3	9	4	5
Total	55	37	18

COMMENT

Thus certain limitations of sympathectomy for hypertension can be established. Hypertension in the adolescent, latent hypertension with increased vascular reactivity, and intermittent hypertension without vascular damage have not been included among surgical indications. Arteriosclerotic hypertension with little or no progression is distinctly a medical problem. Advanced essential hypertension with irreversible damage that is mostly renal does not respond favorably to sympathectomy. While our mortality has stayed below 1% and postoperative complications have been limited to the first six to eight weeks, mostly in the form of intercostal neuralgia,[26] no one should undertake a major, two stage procedure with a one month hospital stay and an additional two months of convalescence without reasonable expectation of improvement.

TABLE 2.—*Causes of Failure Following Sympathectomy for Hypertension in 65 (16%) of 400 Patients, 1940-1948*

Cause of Failure	Method of Detection
Psychic	Interview by psychiatrist
Technical	Postoperative sweating test
Renal	Water tolerance test
Adrenal	Insulin tolerance test Water tolerance test Epinephrine eosinophil count

With the advent of the popularity of sodium restriction, potassium thiocyanate, the newer ergot preparations, and vasodilators, together with the recognition of the importance of the doctor-patient relationship [1a] and the avoidance of stressful situations, a number of patients have experienced symptomatic benefit and objective lowering of diastolic pressure. It should be pointed out, however, that the progress of cardiovascular disease is the most important consideration and that a patient's changed attitude toward his hypertension as brought about by skillful psychotherapy does not necessarily reflect itself in a lowered arteriolar tone or an improvement of regional vascular status.[3c]

The effect of sympathectomy on the three basic mechanisms, namely the neurogenic, corticoadrenal, and renal mechanisms, has already been mentioned. Neurogenic vasoconstriction, whether it originates in the central nervous system or reflex, is effectively blocked by sympathectomy in the areas that mediate splanchnic vasoconstriction. Regeneration, particularly from unexcised ganglia outside the sympathetic chain and splanchnic nerves, is possible and technical modification here may have to be considered.[24] However, as shown by Wilkins and his associates,[15] the limited lumbodorsal sympathectomy and splanchnic section of Smithwick effectively blocks the sudden rises of blood pressure, even if basal pressures are not lowered. The splanchnic vasoconstriction affecting the kidney, liver, and spleen in the upright posture is also eliminated. When brain or heart would benefit from sympathetic denervation, the transpleural, thoracic sympathectomy is logical, with avoidance of the lumbar chain because of its hypotensive effect in the erect position.

TABLE 3.—*Correlation of Water Tolerance With Results Obtained by Dorsolumbar Sympathectomy**

Water Tolerance Pattern †	No. of Cases	Results: Excellent	Results: Good	Results: Unsuccessful
1	15	13	2	..
2	42	3	39	..
3	29	..	21	8
4	9	..	1	8
6	5	..	5	..
Total	100	16	68	16

* de Takats, G., and Fowler, E. F.: Water Tolerance of Hypertensive Patient: Its Relation to Operability, Am. Heart J. **38**: 234, 1949.

† Figure 7. No results were determined for patients with water tolerance pattern no. 5.

Knowledge regarding the effect of splanchnicectomy on the adrenal cortex is meager. Histologic sections, reviewed with the late R. H. Jaffe, suggested marked lipid storage after splanchnicectomy in the dog.[27] Since epinephrine mobilization is decreased following splanchnicectomy, the effect of epinephrine on the pituitary-corticoadrenal axis is decreased and thus a better reserve may be built up. Such a possibility is suggested by the improved eosinophil response of persons with hypertension following splanchnicectomy (fig. 4). This change, together with an increased sensitivity to insulin, comes on slowly over a period of months or years and may explain the late gradual improvement following surgical treatment for hypertension. The influence on the adrenal factor by sodium restriction, potassium thiocyanate, or propylthiouracil [28] explains the beneficial effect of these measures after sympathectomy. Naturally actual reduc-

25. de Takats, G., and Fowler, E. F.: Water Tolerance of Hypertensive Patient: Its Relation to Operability, Am. Heart J. **38**: 234, 1949.

26. Fowler, E. F., and de Takats, G.: Side Effects and Complications of Sympathectomy for Hypertension, Arch. Surg. **59**: 1213 (Dec.) 1949.

27. de Takats, G., and Cuthbert, F. P.: Effect of Suprarenal Denervation and Splanchnic Section on Sugar Tolerance of Dogs, Arch. Surg. **30**: 151 (Jan.) 1935.

28. de Takats, G.: The Phenomenon of Stress in Relation to Human Essential Hypertension, Angiology **2**: 6, 1951.

tion of adrenal tissue may be the optimal procedure, but since the adrenal cortex is one of the cornerstones of defense against stress, such operations must have strict indications unless one wants to encounter a number of listless, nonresistant, vegetable-like persons, not unlike the domesticated rats of Richter that cannot survive in the company of the wild Norwegian rat.[1b]

The idea that renal blood flow is independent of neurogenic control will have to be modified, since the clearance techniques of Homer Smith can be used to test patients in an erect posture after exposure to cold or after a stressful interview. But when basal renal flow is low and since the sympathetics seem to control only efferent arteriolar tone of the glomerulus, the renal factor in hypertension becomes dominant. It is quite possible, of course, that the corticoadrenal factor acts through the kidney, not through a direct vascular effect, and that more will be accomplished in so-called irreversible renal damage by adrenalectomy than has been possible thus far with sympathectomy.

SUMMARY

The initiating and maintaining factors of human diastolic hypertension have been described. The neurogenic, corticoadrenal, and renal factors form a vicious circle, which can be broken at several points, notably by sympathectomy. Sympathectomy, however, has definitely limited indications and has been only undertaken in three groups of patients whose status is readily defined. A five year follow-up of a small number of closely observed patients revealed that improvement in results could be expected mainly by attention to the renal factor. The object of the operation is primarily arrest of cardiovascular disease and not the lowering of blood pressure, which in itself does not mirror organic vascular damage. Operations affecting the increased mesenteric resistance are of most benefit. While newer diets and drugs may replace the necessity for operation in certain persons, sympathectomy has a definite, well-defined place in the treatment of human essential hypertension.

122 S. Michigan Ave.

NATURE AND RECOGNITION OF CEREBRAL PALSY IN INFANCY

Meyer A. Perlstein, M.D.
and
Harry E. Barnett, M.D., Chicago

The diagnosis of cerebral palsy is seldom made in early infancy, despite the fact that in approximately 90% of affected children the condition is congenital.[1] The purpose of this paper is to discuss the nature of cerebral palsy in infancy, with special emphasis on early diagnosis. Much of the information presented is general knowledge, although some observations are reported directly from our personal experiences.

Cerebral palsy is a term used to designate any paralysis, weakness, incoordination, or functional aberration of the motor system resulting from a pathological condition in the motor centers of the brain. The disease may be so localized as to cause only motor symptoms. More frequently, however, the brain damage is diffuse and may also cause convulsions, mental retardation, speech defects, behavior disturbances, and sensory losses of varying degree, particularly in hearing and vision. Sometimes only one of these associated defects exists; oftener, several are in evidence, although one symptom generally predominates.

The diagnosis of cerebral palsy can be established by the experienced clinician in most cases within the first six months of life and in almost all cases within the first year. Early diagnosis is important because it permits early treatment, thus affording better therapeutic results. If the condition is treated from infancy much of the deformity associated with spastic forms of the disease can be prevented. Early diagnosis of cerebral palsy in infants being considered for adoption is important also.

An infant with cerebral palsy may have sufficient undamaged brain tissue to begin developing at an apparently normal pace, not manifesting obvious deviations from the normal until late in infancy. For example, a child with moderately severe damage of the basal ganglions may seem to be developing normally until late in the first year, when he first exhibits athetoid movements and tensions. As a general rule, however, certain stigmas can be recognized in early infancy that indicate the presence of such organic brain disease.

ETIOLOGY

In cases of suspected organic brain damage, a carefully taken history may be more valuable than the physical examination for providing positive clues as to the cause, type, and severity of the neurological disturbance; this is especially true for infants. The history should provide a complete account of the pregnancy, the labor, and the infant's condition immediately after birth. The importance of these factors is underlined by the statistically significant correlation between certain etiological factors and specific clinical syndromes. Some of the more common neurological sequelae of various etiological factors are shown in the following tabulation.

Prematurity	Spastic paraplegia
Breech delivery	Athetoid or spastic paraplegia
Toxemia of pregnancy	Spastic hemiplegia or quadriplegia
Birth trauma	Spastic hemiplegia or quadriplegia
Anoxia	Athetosis
Rh factor and kernicterus	Athetosis, with deafness and paralysis of supravergence
Maternal rubella	Spasticity, with deafness or auditory aphasia, cataract, and congenital heart disease
Precipitate or cesarean delivery	Spastic quadriplegia, ataxia, or rigidity
Placenta previa and abruptio	Athetosis

From Michael Reese and Cook County hospitals.

1. Report of New York State Joint Legislative Committee to Study Problem of Cerebral Palsy, Legislative Document no. 55, Albany, N. Y., 1949.

These indicate more fundamental correlations: brain damage caused by anoxia is usually followed by extrapyramidal syndromes; brain damage caused by primary hemorrhage or trauma is usually followed by pyramidal syndromes.

CLASSIFICATION

Although no two persons with cerebral palsy are exactly alike in the character or degree of motor involvement, it is possible to group most patients according to one of several classifications. These may be based on etiological factors, sites of the neuropathological condition, localization of extremity involvement, severity of involvement, or qualitative nature of motor involvement. A classification, such as the following one, based primarily on clinical symptoms, is the most practical:

1. *Spasticity:* with involvement of the pyramidal tract. These are characterized by stretch reflexes, increased muscle tone, and the classical findings of "upper motor neuron" involvement.
2. *Hyperkinesias:* with involvement of the extrapyramidal tract.
   - A. *Athetosis:* characterized by involuntary, incoordinate, uncontrollable, purposeless movements with varying degrees of muscle spasm or tension.
   - B. *Dystonias:* characterized by marked tension, with greater involvement of the axial muscles and resultant tonic postural attitudes.
   - C. *Tremors:* characterized by uncontrollable motions, pendular or pattern-like in nature, involving alternate agonist-antagonist muscle actions.
3. *Rigidities:* characterized by a resistance to slow passive motion, which is usually intermittent, and by simultaneous contractions of agonist and antagonist muscle groups.
4. *Ataxias:* due to involvement of the cerebellum and its tracts. These are characterized by loss of balance sense, hypotonia, and dyssynergia.

Spasticity and ataxia usually occur as pure syndromes. Hyperkinesia and rigidity frequently coexist, with variable and intermittent predominance of one of these extrapyramidal syndromes. This paper will be concerned primarily with spastic and athetoid cases, which constitute about 90% of all cases of cerebral palsy.

ASSOCIATED HISTORY AND SYMPTOMS

In addition to the prenatal and birth data earlier mentioned, the history should include information on growth and developmental progress, feeding and sleeping habits, reaction to various forms of stimuli, and the presence or absence of convulsions. Frequently parents will exaggerate, or be otherwise unrealistic about, their child's progress and abilities. Normal siblings provide comparative developmental controls for determining the patient's rate of progress.

In the following sections are discussed the symptoms that usually cause the parent to bring a child with cerebral palsy to the physician.

Convulsions.—Convulsions occur in only 0.5% of the general population. In contradistinction, they occur in approximately 40% of children with cerebral palsy, the incidence being about three times as great in spastic children as in athetoid children. A study of 1,000 consecutive patients with cerebral palsy encountered in a pediatric neurologic service revealed that 50% of all spastic patients and 15% of all athetoid patients had convulsive episodes. The seizures in the cerebral palsied child are predominantly of the grand mal type, although petit mal and akinetic or myoclonic seizures are not uncommon. Seizures are often focal in nature. Psychomotor spells in children are not common. As a general rule, the prognosis is worse when convulsive seizures are part of the clinical picture. The nonrecurrent convulsions of the first weeks of life or the febrile convulsions of the first two or three years do not carry the same poor prognosis as the recurrent nonfebrile convulsions of the postneonatal period.

Feeding Difficulties.—Cerebral palsied children often have severe feeding difficulties. This is especially true in those of the athetoid group. The cause of the difficulty may be one, or a combination of, the following factors:

- A. Persistent vomiting, of central or peripheral origin.
- B. Difficulty in swallowing, due to persistence of reversed tongue movements.
- C. Gagging from "swallowing" tongue during feedings or from difficulty with glottis.
- D. Constipation resulting from loss of abdominal muscle tone, increased water loss, and decreased fluid intake.
- E. Excessive gas formation and "colic" associated with constipation.
- F. Spasms of muscles of swallowing and breathing.
- G. Psychogenic anorexia due to forced feeding.

A child with a brain injury resulting in hyperkinesia has increased caloric and fluid requirements, fulfillment of which may be thwarthed by feeding difficulties, thus establishing a vicious circle. These infants may show any or all of the symptoms usually associated with the "hypertonic infant," namely, irritability, failure to gain weight, vomiting that may simulate that of pyloric spasm, colic, constipation, poor feeding ability, excessive crying, back arching, and drawing up of the legs. When a child presents the above picture without apparent cause, and when the symptoms persist in spite of treatment, one should suspect organic brain disease, most likely of the extrapyramidal type.

Drooling.—Normal children begin to drool at the age of 3 months, and may continue to drool for approximately nine months. Drooling in children of this age group, unless excessive and persistent, is not indicative of cerebral palsy. Excessive drooling in cerebral palsied children may be the result of difficulty in swallowing, difficulty in keeping the mouth closed, or reversed tongue movements.

Delayed Speech.—A normal child babbles at the age of 6 months and speaks words at about the age of 1 year. An infant with cerebral palsy may have any type of speech disorder, ranging from complete absence of babbling or language, persistence of babbling, or difficulty in word formation or in language concepts, to minor rhythmical or functional disturbances.

The muscles of the tongue, palate, or larynx, or other muscles of respiration, chewing, and swallowing may be involved in speech impairment of motor origin. The muscle involvement is similar to that found in the extremities of the same patient.

Deafness, partial or complete, and mental retardation may delay or distort the normal speech pattern. Lack of motivation for speech as a result of overindulgence by parents may also delay speech. The syndromes of congenital motor aphasia or of difficulty in language perception or symbol comprehension must also be considered when speech is delayed.

Dental Problems and Teeth Grinding.—Severe metabolic disturbances caused by cyanosis or birth injuries may interfere with the development of enameloblasts in the deciduous teeth. The resulting enamel defects appear in the newly erupted teeth and may be responsible for the rapid erosion produced by the superimposed factor of excessive grinding. Teeth grinding is commoner and severer in cerebral palsied children than it is in normal children. Teething occurs at the same age in both.

Disturbance in Sleep Pattern.—A child with brain damage may be apathetic or hyperirritable. He may sleep almost constantly and may have to be aroused for feedings. On the other hand, if a painful stimulus such as abdominal colic exists, the infant may cry almost constantly and obtain little sleep. Hyperirritability to sound and other stimuli may result in startle reactions during sleep and drowsy states, as well as during the waking hours.

Delay in development of functional activities will be discussed in detail under examination of the motor system.

Sensory defects and mental retardation are not forms of cerebral palsy unless accompanied by signs and symptoms of abnormal motor function. They are discussed in detail in a special section of this paper.

PHYSICAL EXAMINATION

General Observations.—Cerebral palsied children generally appear small for their age. Although properly proportioned, they are often constitutionally smaller and thinner, frequently weighing only 14 or 15 lb. (6.4 or 6.8 kg.) at the age of 1 year. This is especially true of athetoid children. Spastic children, especially of the paraplegic type, may be average in weight or overweight. Tissue turgor may be poor because of increased food demands, impaired feeding ability, and excessive perspiration. Vitamin and caloric requirements are also increased, so that deficiencies, particularly avitaminosis B, frequently occur. Thermolability, particularly the tendency to hyperpyrexia, with or without apparent cause, frequently accompanies extrapyramidal lesions.

Facial Appearance and Expression.—A normal baby begins to smile when 4 to 6 weeks old, and at the age of 3 months smiles frequently when pleasantly stimulated. As he grows older he also develops a differentiated smile, with variation in emotional content depending on the nature of the stimulus. Failure to smile, especially on stimulation, and failure to respond to the examiner's and mother's beckonings by changes of facial expression may be due to mental retardation or to cerebral palsy involving the facial musculature. Spastic infants and those with postencephalitic rigidities may have masked facies, with apparent apathy and failure to respond to stimulation with appropriate facial expressions. Athetoid infants may have very expressive facies, with excessive grimacing and facial distortions that are often inappropriate and unrelated to the specific stimuli. In older infants, attempts to talk or to perform any voluntary movements that increase tension may result in a typical open-mouthed expression ("opera singer's mouth") caused by a downward pull of the mandible.

Abnormal Skull Contour.—Hydrocephalic children are frequently spastic. Internal hydrocephaly usually results in a dolichocephalic head, whereas external hydrocephaly usually results in a brachycephalic head. Asymmetric flattening of the occipital and parietal bones is not rare in cerebral palsied children. The presence of hydrocephaly or other skull abnormalities in themselves do not predicate the coexistence of cerebral palsy. Hydrocephalic children may have an ataxia caused primarily by the enlarged size and weight of the head.

EXAMINATION OF MOTOR SYSTEM

Examination of the motor system should begin with observation of the movements of various parts of the body at rest, on stimulation, and in various positions. The infant should be as relaxed as possible. This is often facilitated by placing him in a prone position or by examining him while he is held by his mother.

It must be reemphasized that a child with organic involvement of the motor centers may show no gross spontaneous evidence of motor abnormality for the first 12 to 18 months after birth, except for delay in general motor development. The severity of signs and symptoms and the age at which they appear are determined by the localization and extent of the organic defect. With pyramidal tract damage, the stigmas may be present immediately at birth or soon afterward, and almost always appear before the age of 6 months. In extrapyramidal tract damage, it may be difficult to make a definite diagnosis before the second six months of life.

In pyramidal tract involvement (spasticity) the lower extremities are affected more severely or exclusively; in extrapyramidal tract lesions (athetosis, rigidity, and tremors) the head, neck, and upper extremities are usually more severely involved. In hemiplegia, over 95% of the cases are of the spastic type, and the upper extremity is usually more severely involved.

The correlation, as shown in the following tabulation, between localization of motor involvement and neurological diagnosis is constant enough to be of help in establishing a specific diagnosis in many cases of cerebral palsy.

Hemiplegia	Spasticity
Paraplegia	Spasticity
Quadriplegia with greatest involvement in upper extremities	Athetosis
Quadriplegia with greatest involvement in lower extremities	Spasticity

Athetosis.—In a normal infant arm and leg movements, such as leg kicking and arm flinging follow a purposeful pattern and are repetitious and predictable in nature. In children with extrapyramidal brain damage arm and leg movements are variable, unpredictable, apparently purposeless in nature, and follow no pattern.

If not spontaneously present, athetoid movements often may be elicited by primary voluntary motions, which then produce secondary overflow athetoid movements in other parts of the body. In tension athetoid children, development of extraneous movements of the lower extremities and trunk on attempted voluntary use of the upper extremities is often found.

Characteristic tensions may develop in the athetoid infant before athetoid motions develop. These tensions may be observed as intermittent stiffening spells or extensor spasms and are often caused by minimal stimuli. In less severe forms the tension may be manifested in straightening of the arms with clenching of fists, or extension of fingers on reaching for objects. The athetoid child may also maintain an attitude of extension at the shoulders and elbows and pronation of the forearms (pronator sign) because of increased muscle tension.

Athetoid movements, usually not manifest for many months, occasionally may be present at birth, as in the so-called congenital athetoid.

Spasticity.—An infant with spasticity may reveal evidence of such involvement in the first few months of life. In a quadriplegic infant there is a diminished amount of total motion, and the motion that is present is explosive and jerky. The child frequently presents an attitude of flexion of the upper extremities and extension of the lower extremities and often assumes characteristic reflex positional attitudes of the trunk and extremities. In infants with spastic hemiplegia, some voluntary motions may be possible in the involved extremities, but they are poorly performed. There is a disinclination to use the involved limbs, particularly the arm, disproportionate to the observable motor disability. In spastic paraplegia the upper extremity function is normal, but patterns of kicking, reciprocation, and other lower extremity functions are abnormal and are delayed in appearing.

A tendency to hold the thighs adducted because of spasticity of the adductor muscles exists in the presence of most pyramidal tract lesions. This "scissoring" tendency usually increases, and may become very noticeable when the child is held erect or when it attempts to stand and walk. It is usually accompanied by ankle equinus and internal rotation at the hip joints. In these children, the hips and knees are usually held in rigid extension on supported standing. Scissoring may also occur in athetoid children but, in contrast to that seen in spastic children, it is less constant and usually does not result in contractures.

If held by the legs and lowered until the head touches the floor, a normal baby will drop his arms as if to protect his head. When an infant with spastic upper extremities is so held, his arms may not drop but may be held rigidly in front of the body with extended elbows and pronated forearms, or only one arm may drop (hemiplegia), depending upon the localization of motor involvement. This may also occur in athetoid infants.

Occasionally, transient forms of spasticity, especially spastic hemiplegia, may occur. They are usually associated with convulsions, and often are due to intracranial hemorrhage or vasospasm. All the signs and symptoms of spasticity may be present at the age of 2 or 3 months and may disappear completely by the age of 1 year.

SPECIFIC MOTOR TESTS AND OBSERVATIONS

Stretch Reflexes and Resistance to Passive Motion.—The exact type of neurological involvement may be diagnosed in a high percentage of cases of cerebral palsy by tests designed to elicit specific properties of the affected muscles. A positive stretch reflex may be considered pathognomonic of a spastic muscle. Typically, a spastic muscle may be put through a full range of motion passively, if the movement is carried out slowly. As the speed of the movement increases, a point is reached at which resistance is encountered. If the passive motion is rapid, it is quickly halted by a strong muscular contraction of the stretched muscle. This latter action is termed the stretch reflex. In mild cases, or in those accompanied by weakness or hypotonia, it may not be elicited until after the first year, but in severe cases it may be present within the first month of life. The stretch reflex is most frequently encountered in the antigravity muscles: the hip adductors, triceps surae, and quadriceps. It may also spread to the contiguous muscles and produce an involuntary overflow or mass motion.

Ankle clonus is a form of sustained stretch reflex and is more easily elicited when the patient is lying on his abdomen with his knees flexed. A tension athetoid patient may also have ankle clonus. However, with relaxation the ankle clonus of a spastic child becomes more evident and more sustained, whereas that of a tension athetoid child usually disappears.

Reaction to varying speeds of motion can also be used in testing muscles affected by extrapyramidal lesions. In a tension athetoid patient slow movements at a joint are usually resisted intermittently throughout the range of motion, whereas rapid movements will encounter little, if any, resistance. A simple method of differentiating tension athetosis from spasticity is by attempting to shake an extremity, e. g., the wrist and fingers, with rapid motion. An athetoid limb will begin to flail; a spastic limb will become tense.

The patient with rigidity shows constant or intermittent resistance to slow movement of his limbs; these reactions have been given the descriptive names of "lead pipe" and "cog wheel" rigidity, respectively. Either reaction is elicited by slow movement at the major joints and is overcome by rapid movement.

Mass Movement.—The infant with organic brain disease, either pyramidal or extrapyramidal, may display mass movements of a fetal nature in which all the extremities may be extended or flexed, or some flexed and some extended simultaneously, as in the tonic neck reflexes. These are reflex motions and usually assume the pattern of a protective action against bodily harm. They occur when the child is frightened or when strenuous attempts are made at voluntary movements. Although the movement is initiated in one extremity it may spread to all four extremities. These children may also have forceful arching tendencies of the head and back.

Reciprocal Leg Movements.—A normal child has a reciprocal kicking pattern at birth. When supported in an erect position with feet on the table a child may make walking movements immediately after birth. A delay in development of these reciprocal movements may be the first sign of brain damage. Such delay is much commoner

in pyramidal tract disease. In the absence of evidence of mental retardation, failure of reciprocation to appear by 3 months of age may be considered presumptive evidence of cerebral palsy.

Head Support and Motion.—Normally an infant in the prone position is able to lift its head within a few weeks after birth, and in the supine position is able to do so by the age of 12 weeks. Inability to support the head in the prone position at 6 to 12 weeks should cause suspicion of organic brain damage. Head drop in the supine position after the age of 4 months, even in the presence of good extension power in the prone position, is suggestive of cerebral palsy. Back and forth flopping motions of the head or rolling head motions in an erect attitude may also be signs of organic brain disease.

Prehensile Activities.—Upper extremity function may be tested at an early age by observing the manner in which a child responds to objects in a predetermined testing situation. Gesell and Amatruda,[2] and others have described what a normal child will do with objects placed before him or into his hand; for example, an 8-week-old child sitting in a high chair will retain an object placed in its hands for one to two seconds before dropping it; at the age of 20 weeks he will reach for and grasp objects such as rattles and cubes with one or both hands; at 40 weeks he is able to transfer a cube from hand to hand and to hold two cubes as if to match them; at 1 year he attempts to place one cube upon another, grasp small objects with almost adult precision, and release objects on request.

Cerebral palsied infants with upper extremity involvement may be long delayed in development of prehensile skills, because of lack of voluntary movement, incoordination, or a persistence of hypertonic attitudes of the fingers and wrists. Normally an infant maintains his fingers in a clenched position for the first 8 to 12 weeks. Thereafter, they are held extended or slightly flexed, and on attempted grasp the fingers gradually flex as they approach an object. In a spastic infant the hand may remain clenched even as the object is being fingered, and his attempts to open the hand may be jerky and incomplete. In athetoid infants the objects may be approached with an extended or flexed wrist and with clenched or extended fingers. The greatest difficulty may be in grasping, rather than in releasing, objects. A spastic child will reach and grasp with characteristically slow, measured, and awkward movements, but he will generally move the hand in a straight line toward the object. An athetoid child will reach and grasp in a rapid, erratic, excited manner, with many tremors and undulations of the extremity as the object is approached. Frequently the object is missed entirely.

In hemiplegic infants the difference in motor ability between the two hands may be obvious within the first few months of life. It may be demonstrated by placing a towel over the infant's face. Normally he will quickly attempt to remove it with one or both hands. By restraining one hand, the functional ability of the other hand can be observed as the child attempts to displace the towel from his face.

Upon careful observation it may be noted that the child with motor involvement of the upper extremities will display eagerness and determination to perform a task with his hands and, if helped to accomplish it, will manifest satisfaction. He may "grasp with his eyes" before he is able to grasp with his hands. The interest shown in reaching for objects and the concentration and perseverance displayed in performing test acts are of importance in differentiating between poor performance due to motor disability and that due to mental retardation.

Sitting, Standing, Crawling, and Walking.—The normal rate and pattern of development of sitting, crawling, creeping, standing, and walking have been previously described by others in great detail. A knowledge of such normal functional behavior in infants is necessary for physicians or psychologists testing children for motor disability or mental retardation. At the age of 24 weeks a normal baby can roll from the supine to the prone position. At 28 weeks he begins to sit alone, erecting his trunk for a brief moment and then leaning forward onto his outstretched palms. At 40 weeks an infant sits unaided with good postural control and is able to go from the prone to the sitting position; at this age he is able to get up on his hands and knees, to creep forward, to pull himself to his feet while holding onto a railing, and to support his his entire weight on his feet when his hands are held. At 12 months a baby can walk with one hand held. At 15 months he can walk alone and attain the standing position unaided.

An infant with cerebral palsy may be completely unable to perform any of the above functions, may be late in performing them, or may perform them in a manner characteristic of the type of motor disability.

Sitting may be delayed for many months in mild cases and for years in severe cases. It may be characterized by a persistence of the fetal flexion attitude, by extensor spasms of the back muscles, increase of athetoid motions, or increase in muscle tonus. Balance is easily upset. In older children "knee-sitting" commonly occurs and results in typical deformities of the lower extremities.

Crawling and creeping are also delayed and may be accomplished by alternating frog-like motions of the arms and legs, rather than by reciprocal motions. This is commoner in spastic children. An infant with severe hand involvement, especially the athetoid child, may skip the crawling and creeping stage and go directly from sitting to walking. Occasionally, a severely disabled child may first learn to locomote on his back, using his legs to push his head and trunk forward.

Standing and walking are usually delayed in children with cerebral palsy. In a mild spastic hemiplegic child of normal intelligence they may be delayed only two to four months. A mildly athetoid child may walk with only 6 to 12 months' delay. In a moderately severe spastic paraplegia walking may be delayed one to two years. A severely athetoid child may not be able to walk unaided until the age of 6 or 7 years, if ever. In spastic children attempts to walk are characterized by scissoring and flexion at the hips, flexion at the knees, and equinus at the ankles. The movements are initiated slowly and performed awkwardly. In athetoid children attempts to walk

2. Gesell, A. L., and Amatruda, C. S.: Developmental Diagnosis: Normal and Abnormal Child Development, Clinical Methods and Pediatric Applications, ed. 2, New York, Paul B. Hoeber, Inc., 1947.

may cause an increase in muscle tension or in athetoid movements of the lower extremities, with an overflow of motion to the trunk and upper extremities.

The pattern of functional deformities in the spastic child maintains a rather constant picture in a given case, varying little from step to step and from day to day. In contrast to this, the athetoid child usually shows noticeable daily variations in the pattern of functional deformities. Ankle valgus or varus, knee flexion or extension, and hip adduction or abduction may be observed in the same athetoid child on repeated examinations. In quadriplegic children there is loss of arm reciprocation in walking.

The degree of functional retardation or disability associated with cerebral palsy is more dependent on the severity than on the type of the motor disability. A mildly athetoid child may progress more rapidly than a more severely spastic child, although a spastic child usually develops more functional ability in an extremity than does an athetoid child having a comparable degree of involvement. The spastic child usually improves more rapidly with therapy.

Tongue Motions.—Observation of tongue motions in an infant may provide one or more pathognomonic signs of cerebral palsy. Forward pushing motion of the tongue (elicited by putting a solid object into an infant's mouth), which is normally reversed at the ages of 4 to 6 weeks, may persist for months or years, especially in athetoid children. There may be difficulty in lifting the tip of the tongue and in performing lateral tongue motions. The child should be observed when crying to see if the tongue tip is normally lifted and cupped. If an object is placed against the mucous membrane of the cheek, a normal child will initiate a movement of the tongue to that side. In cerebral palsied children there may be no such lateral tongue motions. The tongue may be held in a retracted state. A stretch reflex in the tongue, reported by other investigators, has not been observed in our cases.

Eye Movements.—Examination of the extrinsic muscles of the eye may produce presumptive evidence of cerebral palsy. Strabismus, nystagmus, and paralysis of gaze (loss of conjugate movements) are likely to occur with organic brain damage. Strabismus associated with cerebral palsy is characterized by an extreme variability in the degree of squint observed in a patient on repeated examination. Internal strabismus is much commoner than the external form. Nystagmus associated with poor vision caused by optic atrophy or congenital eye defects is usually of the pendular type with no slow or rapid components, but with movements that are coarse, irregular, and "searching" in nature. Nystagmus occurring secondarily to organic brain damage is usually described as jerky, with slow and fast components.

Paralysis of gaze, especially of supravergence and infravergence, is frequently associated with the Rh type of athetosis. These motions may, however, be elicited by "synkinetic" movements, which may be defined as involuntary movements or reinforcement of voluntary muscle action due to a voluntary movement of another muscle or in response to a reflex. In looking up or down, these patients will usually extend or flex the neck. In following objects, an infant with cerebral palsy may also have obvious difficulty with his associated head and neck movements, and he may display a "surprised" look, with elevation of the eyebrows and eyelids.

Contractures.—Contractures due to muscle action rarely occur before the age of 1 year, but they may occur earlier in cases of severe spasticity; later, they are commoner in spasticity than in athetosis. The flexors of the ankles, hips, and knees, and the hip adductors are most frequently involved. Contractures in the newborn infant may indicate arthrogryposis multiplex congenita.

• *Atrophy.*—Severe atrophy of muscles due to cerebral palsy is rare during infancy. If present in one extremity, another type of lesion such as Erb's palsy or poliomyelitis must be considered. In infants with generalized muscle atrophy a diagnosis of amytonia congenita must be considered; however, in infants with congenital hemiplegia there may be a grossly obvious lack of extremity development, rather than atrophy, of all tissues of the involved limbs. Atrophy and shortening are usually more noticeable in the upper than in the lower extremity.

Atonic Muscles.—A spastic or athetoid child may have a predominance of atonic or hypotonic muscles, and a subclassification such as atonic spastic diplegia and atonic athetoid quadriplegia can be made. The hypotonia may be due to a cerebellar lesion or a cerebellar tract lesion superimposed on the pyramidal tract or extrapyramidal tract involvement. Atonic spasticity is frequently diagnosed as muscular atrophy (amyotonia congenita) or muscular dystrophy. In contrast to patients with the latter diseases, however, the atonic spastic patient usually has normal or increased deep-tendon and stretch reflexes and has a greater degree of muscle power on voluntary contraction than one would expect from examination of the resting muscle. Typically, on lifting a patient with amyotonia congenita by the armpits, the lower extremities remain extended, whereas on lifting an atonic spastic patient in the same way, the hip and knee points usually assume a flexion attitude.

Isolated muscles or muscle groups that are weak or that have lost all voluntary power may be found. These are seen most frequently in spastic patients, but do occur in athetoid patients, and have been variously termed "0 C." (zero cerebral), flaccid, or mentally alienated muscles. They are found most frequently in direct opposition to the more severely spastic muscles and are usually found to be overstretched by contractions or contractures in the more powerful antagonists. They are not truly flaccid muscles, for motion can be elicited in them by specific reflex or synkinetic movements designed to innervate or reinforce these muscles; e. g., resisted thigh flexion may elicit contraction of the anterior tibial muscle. This muscle flaccidity may be due to supranuclear lesions similar to those responsible for loss of lateral tongue motions or paralysis of gaze.

REFLEXES

The normal infant shows certain characteristic reflex actions in the first months of life. Among these are the deep tendon reflexes, tonic neck reflexes, the Moro reflex, sucking and rooting reflexes, and the grasp reflex. Absence, persistence, or perseveration of these reflexes may be the earliest evidence of organic brain disease.

Deep Tendon Reflexes.—Deep tendon reflexes are usually normal in athetoid infants and are increased in spastic infants. It should be noted, however, that neurological signs, even in a normal infant, are variable and unpredictable. A normal child may have increased deep tendon reflexes as a result of reinforcement, especially when frightened or crying. To be of value the examination must be performed while the child is relaxed. This is best accomplished by laying the child on its abdomen with its knees flexed. Normally knee jerks are not elicited under these conditions. If present they are probably exaggerated.

Superficial Reflexes.—Superficial reflexes are usually present in all forms of cerebral palsy and are of little diagnostic significance.

Tonic Neck Reflexes.—Tonic neck reflexes are elicited by rotating or inclining the head first toward one shoulder then toward the other. Extension of the limbs on the side of the body toward which the jaw is inclined and flexion of the limbs on the opposite side constitute a positive sign. In a normal child this sign becomes negative before the 16th week of life. Bilaterally positive tonic reflexes after this age are indicative of organic brain damage. Inequality between the right and left tonic reflexes may be due to asymmetric brain damage. When brain damage is severe, these reflexes may persist into adult life (Magnus-de Kleijn phenomenon).

Moro Reflex.—The Moro or startle reflex may be elicited by striking the table on which the baby lies, by startling him with a loud noise, or by sudden release of his outstretched arms. Normally the arms are thrown outward and then are brought toward each other in an embracing motion. The hands are fanned out at first and then clenched tightly. The motions are usually associated with a cry due to fright. The Moro reflex usually disappears by the 16th week of life. Its persistence indicates organic brain damage. It may be absent with severe increase of intracranial pressure or severe damage in the subcortical brain.

Rooting and Sucking Reflexes.—The rooting and sucking reflexes can be discussed together. A normal infant, when one cheek is touched, will turn its head toward the touched side and make sucking motions. These reactions are present from birth. Failure to elicit the rooting and sucking reflexes, and persistence of tongue pushing motions after six to eight weeks of life are presumptive signs of intracranial damage or developmental defect.

Grasp Reflex.—Normally an infant maintains his fingers in a clenched position up to the age of 8 to 12 weeks. If the examiner inserts a finger into each fist of the infant he can often lift it from the bed by this method because of reflex reinforcement of the infant's finger flexion. With abnormal motor function caused by organic brain disease the clenched fist reflex may persist, so that the child may be lifted in the above fashion for many additional months or even years. Other infants with motor involvement, however, may have a poor grasp reflex, as well as a poor release mechanism. The grasp reflex may be absent from birth.

Pathological Reflexes.—Pathological reflexes, such as the Babinski reflex, are not significant in infants, presumably because of incomplete myelinization of the pyramidal tract. Their presence in an infant prior to the age of 18 months is not necessarily indicative of organic brain damage.

HEARING AND VISION

Hearing and vision may be tested in a crude fashion by eliciting blink reflexes with visual and auditory stimuli. The absence of these reflexes may indicate the presence of blindness or deafness, both of which are commoner in cerebral palsied children than in otherwise normal children. However, they may be absent also in patients with severe mental retardation. From three to six months after birth an infant with normal vision and control of eye movements will fixate on a light held before him and will follow the course of the light with a steady fixation. An infant with impaired vision will display an unsteady, wobbly fixation when tested in a similar way. A totally blind infant will neither fixate upon nor follow the course of a light.

A child may present a picture of deafness but have normal hearing associated with a loss of auditory perception. The differential diagnosis between true deafness and auditory aphasia may be made by special tests (e. g., arousal tests with electroencephalography, conditioning tests, and skin resistance tests).

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of organic brain disease in infancy all the following possibilities must be considered:

A. Simple mental retardation.

B. Conditions which may produce the clinical picture of the "hypertonic" infant, such as food allergies, gastrointestinal anomalies, and painful conditions of the extremities.

C. Meningitis and encephalitis—acute and chronic forms.

D. Familial degenerative diseases, such as Tay-Sachs and amaurotic familial idiocy.

E. Endocrine dysfunctions, including hypothyroidism.

F. Isolated sensory defects such as deafness and blindness.

G. Poliomyelitis.

H. Erb's palsy.

I. Arthrogryposis multiplex congenita.

J. Amyotonia congenita.

K. Brain tumor.

L. Debilitation and malnutrition, which may temporarily impede both mental and physical development.

M. Postconvulsive states.

Complete examination of a child suspected of having organic brain disease may require any or all of the following diagnostic procedures:

1. *X-rays of the skull:* For skull fractures, increased intracranial pressure, and calcification, as in toxoplasmosis.

2. *X-rays of long bones:* For bone age, plumbism, and developmental defects.

3. *Complete blood studies* (often on parents as well as on child): May include studies of Rh factor, cholesterol, proteins, calcium, and phosphorus; smears for

basophilic stippling and serologic tests for chronic infections like syphilis and toxoplasmosis.

4. *Spinal or subdural puncture* (in selected cases): For central nervous system infections, increased pressure and subarachnoid block or hemorrhage.

5. *Eyeground examination:* For choked discs, chorioditis, retinal changes, and anomalies.

6. *Electroencephalogram:* May be of value after age of three months in showing paroxysmal patterns, rhythmic disturbances, and asymmetries.

7. *Pneumoencephalogram* (in selected cases): Correlations between clinical and pneumoencephalographic findings are not too great. However, it is of value in corroborating a diagnosis of subdural hematoma, marked cortical atrophy, neoplasms, internal hydrocephalus, and certain anomalies of the brain.

8. *Psychological evaluation:* To determine functional abilities and performance levels of the patient.

MENTAL RETARDATION AND CEREBRAL PALSY

The incidence of sensory defects and mental retardation is greater among cerebral palsied persons than among the general population. Mental retardation without motor involvement may cause a delay in many phases of maturation and motor development, and thus simulate the clinical picture of cerebral palsy. Mental deficiency may be classed as (1) undifferentiated, (2) associated with organic brain disease, or (3) associated with organic brain disease and cerebral palsy.

Our experience, based on a study of 1,000 consecutive cases of cerebral palsy seen in the children's neurology clinic at the Cook County Hospital and in private practice, has indicated that over 35% of all children with cerebral palsy have an appreciable degree of mental retardation, i. e., an intelligence quotient below 70. Mental retardation is approximately twice as common in spastic children (50%) as in athetoid children (25%). Also, there is a high correlation between educability and convulsions in these children. Only 45% of cerebral palsied children with convulsive symptoms were found to be educable, whereas 75% of those without convulsions were classed as educable. It should be noted that cerebral palsied children who are rated as educable may have some degree of intellectual impairment. Without their brain damage, their intelligence quotients might have been higher. This is indicated by the fact that the average intelligence quotient in educable cerebral palsied children is below 100 and is generally less than that of their unaffected siblings.

Standard psychological tests frequently fail to give a true picture of intelligence in children with cerebral palsy because of the associated auditory and visual defects, emotional instability, speech and perceptive difficulties, and the lack of stimulation, motivation, and experience that are often found in children with brain injuries.

The most satisfactory tests for intelligence in infants with cerebral palsy are those based on developmental rates. Tests devised by Gesell and Amatruda,[2] modifications of these, and other tests, such as the Psyche-Cattell, the Vineland Social Maturity Scale, and the Kuhlman test, are well suited for this purpose; they are designed to determine the degrees of motor, social, language, and adaptive behavior development. As a general rule the normal child exhibits parallel progressive rates of development in all four spheres. The same is true of the child with undifferentiated mental deficiency. However, in mental retardation due to organic brain disease the rates of development in the four spheres are usually uneven and disproportionate. Such disparity should lead one to suspect organic brain disease, and, possibly, associated cerebral palsy.

Children with undifferentiated mental retardation are generally normal in appearance and stature, except in cases of Mongolism or multiple congenital anomalies. These are usually good babies, easy to care for and placid; they cry little, sleep well, and have few feeding problems. Their developmental tests show equal retardation in all spheres (speech, social, motor, and adaptive) quantitatively proportionate to the intellectual defect present. These children display little interest in performance tests and little pleasure at being able to perform such tests. Performance of motor tests may be characterized by a clumsiness that might be considered normal in a child of younger age. They may show a failure of or a delay in response to all forms of stimuli (auditory, visual, and pain-producing), and they may fail to respond to parents or environment. Facial expression in such a child is typically apathetic; he fails to smile or has an undifferentiated smile in response to various pleasant stimuli at the age when differentiated responses are normal. In these children the rate of conditioning is slow. There is no evidence of muscle weakness or aberration of muscle function. There is no spasticity, athetosis, or rigidity. The gait may appear ataxic, but the unsteadiness is primarily the result of lack of skill or balance and resembles the gait of a normal child just learning to walk. Convulsions are no more frequent in these children than they are in normal children.

If mental retardation is associated with organic brain disease without cerebral palsy a higher incidence of convulsive episodes and an increased percentage of feeding and behavior problems usually occur. The developmental tests may show uneven performance, although the discrepancies and the relative delay in motor function are not so great as those seen in cerebral palsy.

The diagnosis of mental retardation in the presence of cerebral palsy is difficult and can be made only after careful evaluation of the signs and symptoms referrable to motor dysfunction. Only experience can lead to proper evaluation of the developmental rate in the presence of motor involvement. The expected retardation in motor or speech development must be estimated in terms of the observable clinical motor defects; retardation beyond this estimate may then be attributed to associated mental retardation; for instance, in a child with spastic paraplegia speech development is usually not delayed. The occurrence of such delay should suggest associated mental deficiency.

A child with spastic paraplegia may not be able to walk at a normal age but will have normal hand function. Even though unable to walk he may be able to use a tricycle at the normal age of about 24 months. In contrast to this, a child who is mentally defective and is, therefore, unable to walk will usually be unable to ride a tricycle at

the normal age. In hemiplegic children there is usually only slight delay in walking, especially in milder cases. The occurrence of a long delay in walking (over six months) in a child with mild hemiplegia is indicative of concomitant mental retardation.

A child with cerebral palsy without mental retardation develops normally in all motor functions except those handicapped by his disability.

A spastic or athetoid quadriplegic child may not be able to reach for or grasp objects, because of poor muscle control; nevertheless, he will usually exhibit some concentrated effort, eagerness, and desire to perform these tasks. In contrast to this, a child with simple mental deficiency, without cerebral palsy, will reveal neither ability to perform an act nor desire to attempt it. One should consider intent, desire, and determination, as well as achievement, in assessing the condition of these children.

SUMMARY AND CONCLUSIONS

The diagnosis of cerebral palsy can be made in most cases before the age of 6 months and in almost all cases by the age of 1 year. The early diagnosis of cerebral palsy is important for initiating appropriate therapy in infancy and for providing an earlier, more reliable developmental prognosis.

The diagnosis of cerebral palsy in infancy can be made more readily if the physician is aware of the following factors: (*a*) relationship between etiological factors and types of cerebral palsy; (*b*) developmental rates and patterns in normal and abnormal children; (*c*) frequency of occurrence of associated defects and specific symptoms; (*d*) specific tests designed to elicit abnormal muscle properties and motor functions; and (*e*) the differential diagnosis between mental retardation and cerebral palsy.

116 S. Michigan Ave. (Dr. Barnett).

FACTORS RELATING TO THE DISTRIBUTION OF PHYSICIANS

H. G. Weiskotten, M.D., Skaneateles, N. Y.

Approximately 30 years ago, soon after taking over the deanship of Syracuse University College of Medicine, I became interested in what became of the graduates of our schools after graduation. Beginning with the graduates of 1915, I initiated a study of every fifth class. Insofar as possible, studies were made for these classes of the location and nature of practice of the graduates of all the medical colleges of the United States and Canada six years after graduation. The studies were based on questionnaires sent to every physician.

The results of studies of the graduates of 1915, 1920, 1925, and 1930 have been published as a series of reports in the *Journal of the Association of American Medical Colleges*. Because of the impact of World War II on the physicians of the country, it was considered unwise to attempt in 1941 a study of the graduates of 1935 or in 1946 a study of the graduates of 1940. Hence, it was not until 1950—fortunately before the Korean War started—that a study of the graduates in these two years was undertaken. To date, the combined studies have involved a total of 24,934 questionnaires, of which 72.1% were filled out and returned.

The present study was supported by a grant from the United States Public Health Service. The studies deal with a variety of factors relating to medical education and the practice of medicine, such as age at graduation, internship and residency training, location for practice, nature of practice, specialization, specialty board certification, and full-time and part-time salaried positions.

The questionnaires were returned by 65.7% of the graduates of 1935, and 72.3% of the 1940 graduates of American medical colleges. Each physician who did not return his questionnaire was looked up in the 1950 edition of the American Medical Directory, and the information given there was used to determine how representative the questionnaires returned could be considered. The results appeared to warrant the general acceptance of the tabulations from the questionnaires returned.

I believed that it might be of interest if I presented as a preliminary report a few of the over-all findings of the present study relating to the distribution of graduates. It is hoped that a full report of all aspects of the study can be published in the near future.

The distribution of physicians is something like the weather. There is much talk about it and it has appeared that there is little we can do about it. Efforts to "seed the clouds" over arid areas seem to offer little promise. It is obvious that there are many factors involved in the location of physicians in this country. Although some of these are measurable, others are so personal and varied that it appears to be hopeless to attempt to measure them. In this preliminary report, I will present certain data relating to the group as a whole. In the final report data will also be presented for the individual schools.

Table 1 shows the distribution of the graduates in the country in which they are practicing. It indicates that almost all of the graduates of American medical colleges practice in the continental United States, as do approximately 25% of the graduates of the Canadian medical colleges. Half of the graduates of 1935 and three-fifths of the graduates of 1940 of the Canadian colleges practicing in the United States lived in the United States before entering medical college. At the same time it should be recognized that the Canadian colleges as a group in their production of physicians have served as the equivalent of another medical college in the United States. Their contributions in 1930, 1935, and 1940 were respectively 66, 58, and 73 graduates.

The distribution of the graduates practicing in the various states of the United States in relation to population distribution and per capita income was tabulated. In an effort to minimize the criticism of adhering to state lines

Read before the Forty-Eighth Annual Congress on Medical Education and Licensure, Chicago, Feb. 11, 1952.

Miss Marion E. Altenderfer of the United States Public Health Service analyzed the questionnaires and prepared the tables.

as well as to simplify the presentation at this time, I am presenting these data in terms of the recognized nine geographic regions of the states (table 2). While many other factors are involved, the table appears to indicate that, in general, the regions of the country with higher per capita incomes tend to attract a percentage of graduates equal to, or greater than, the percentage of the entire population that resides in the area. Those with lower per capita incomes fail to attract their full quota on a population basis. More detailed study of the individual states indicates that, depending somewhat on their per capita incomes, states in which there are no medical colleges fare about as well in the distribution of graduates on a population basis as the states with one or more medical colleges.

TABLE 1.—*Percentage Distribution of 1925, 1930, 1935, and 1940 Graduates of American and Canadian Medical Colleges by Place of Practice*

	American Colleges				Canadian Colleges			
Place of Practice	1925	1930	1935	1940	1925	1930	1935	1940
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Continental U. S.	99.0	98.1	98.6	98.5	39.0	24.3	24.5	24.8
Canada	*	0.4	0.1	*	59.6	71.3	73.4	73.2
U. S. Territories	0.8	0.5	0.8	0.7	0.4	.....	.....	.....
Other	0.1	1.0	0.5	0.7	1.0	4.4	2.1	2.0

* Less than 0.1%.

The size of the communities in which recent graduates locate has attracted considerable attention. Table 3 shows the percentage distribution of the graduates of 1930, 1935, and 1940 in private practice by the size of the communities in which they are located. Over the period covered by the study there appears to have been a slight decrease in the proportion of graduates practicing in cities of 500,000 or more population and a slight increase for cities of 100,000 to 500,000 population. There has been an increase in the percentage locating in communities of 25,000 to 100,000 population. There has been a definite decrease in the proportion practicing in communities of less than 5,000 population.

TABLE 2.—*Percentage Distribution of 1935 and 1940 Medical College Graduates Practicing in Each Region, Percentage of 1950 Population in Region, and per Capita Income*

Region	Graduates, %		Population in	Per Capita Income,
	1935	1940	1950	1950
United States	100.0	100.0	100.0	$1,436
Middle Atlantic	26.6	21.8	20.0	1,727
Pacific	12.2	14.8	9.6	1,710
East North Central	19.2	18.2	20.2	1,603
New England	6.8	8.1	6.2	1,558
West North Central	7.3	8.4	9.3	1,387
Mountain	4.3	3.5	3.4	1,362
West South Central	6.8	7.8	9.6	1,145
South Atlantic	12.9	12.7	14.1	1,139
East South Central	3.9	4.7	7.6	867

As might be expected, there was considerable variation in the proportion of graduates of the different medical schools practicing in the smaller communities. For example only 5% of the graduates of Johns Hopkins University School of Medicine were practicing in communities of less than 10,000 population as contrasted with 50% of the graduates of the University of Vermont College of Medicine.

The proportion of graduates practicing in the same city in which the medical college they attended is located has remained relatively constant for the country as a whole. The proportions of the 1930, 1935, and 1940 graduates who practice in the same city as their medical college were 18.8%, 19.6%, and 18.5%, respectively. However, there are not only marked differences between the distribution of graduates of the different colleges, but also definite and marked changes in trend on the part of the graduates of certain medical colleges. For example, there has occurred a marked progressive decrease in the proportion of the graduates of Columbia, Cornell, Long Island, New York Medical College, New York University, Marquette, and Johns Hopkins who practice in the same city in which the school from which they graduated is located. The studies show, however, that New York University School of Medicine still had the highest proportion of graduates in 1930, 1935, and 1940, who practice in the city in which the college is located or 77%, 58%, and 51%, respectively.

TABLE 3.—*Percentage Distribution of 1930, 1935, and 1940 Graduates of American Medical Colleges in Private Practice by Size of Community*

Size of Community	Year of Graduation		
	1930	1935	1940
Total	100.0	100.0	100.0
500,000 and over	28.8	25.6	24.2
100,000-499,999	16.1	20.8	21.5
50,000-99,999	6.8	9.7	10.9
25,000-49,999	5.6	9.3	11.9
10,000-24,999	9.2	11.6	10.9
5,000-9,999	7.5	6.7	6.6
2,500-4,999	25.6*	5.0	4.6
Under 2,500	.....	10.7	8.8
Unknown	0.4	0.6	0.6

* This is the total for all communities of less than 4,999.

TABLE 4.—*Percentages of 1930, 1935, and 1940 Graduates of Public and Private American Medical Colleges Practicing in Same State as Medical College Attended*

Form of Medical College Control	Year of Graduation		
	1930	1935	1940
Total	50.5	49.7	44.3
Public	49.3	53.6	50.4
Private	51.2	47.5	40.6

As the studies have progressed, there has been an apparent decrease in the proportion of graduates who are practicing in the same state as the one in which their medical college is located. Table 4 gives the percentage for the graduates of the publicly and privately supported

medical colleges, which indicate that the decrease can be attributed to the graduates of the privately supported schools. In extending the location of the graduates in relation to the location of the medical college to a broader area, our tabulations show that 62.6% of the 1935 graduates and 57.5% of the graduates of 1940 practice in the same region as the one in which their college is located.

In the study of the 1935 and 1940 graduates the relationship of the previous residence of the student to the location of the medical college attended and the ultimate place of practice was studied. Table 5 shows for the publicly and privately supported medical colleges the percentage of graduates who lived in the same city, the same state, and the same region as their college before entering medical college.

TABLE 5.—*Percentage of 1935 and 1940 Graduates of American Medical Colleges Whose Prior Residence was in the Same City, Same State, and Same Region as the College, by Form of Medical College Control*

Form of Medical College Control	Percentage Whose Prior Residence Was in		
	Same City as College	Same State as College	Same Division as College
Total	25.7	60.5	71.5
Public	25.7	78.1	85.4
Private	25.7	50.3	63.4

TABLE 6.—*Percentage Distribution of 1935 and 1940 Graduates of American Medical Colleges in Private Practice in Various Size Communities, by Size of Community of Prior Residence*

Size of Community of Prior Residence	All Sizes	Size of Community of Practice					
		500,000 and Over	100,000-499,999	50,000-99,999	10,000-49,999	2,500-9,999	Under 2,500
All sizes	100.0	100.0	100.0	100.0	100.0	100.0	100.0
500,000 and over	26.8	64.6	12.1	16.4	15.6	13.3	15.3
100,000-499,999	16.9	6.8	46.8	7.4	10.5	10.5	10.7
50,000-99,999	7.9	4.3	5.0	35.0	4.9	4.4	5.9
10,000-49,999	18.0	9.0	12.7	14.1	39.1	14.6	14.3
2,500-9,999	11.2	4.5	8.0	9.7	10.1	33.9	13.0
Under 2,500	12.5	4.4	8.8	11.4	13.7	16.7	35.6

It is interesting that the percentage who lived in the same city as the college attended is identical for the publicly and privately supported colleges. As would be expected, the percentage who lived in the same state as the college is much higher for the publicly supported schools. With the increased residency requirements for admission recently enforced by many of the state-supported medical colleges, the percentage of state residents graduating in the future from this group of colleges will far exceed those for 1935 and 1940.

Table 6 shows the percentage distribution of graduates of American medical colleges in private practice practicing in the various size communities by the size of community of residence before entering medical college. It is obvious that, for each size of community of practice, a far larger percentage of graduates had their prior residence in a similar size community than in any other size community.

Table 7 deals with the same relation between the size of prior residence and the size of the community of practice from a different viewpoint. This table shows the percentage distribution of those whose prior residences were in the various size communities. It appears to me that the

TABLE 7.—*Percentage Distribution of 1935 and 1940 Graduates of American Medical Colleges in Private Practice Whose Prior Residence Was in Various Size Communities, by Size of Community of Practice*

Size of Community of Prior Residence	All Sizes	Size of Community of Practice					
		500,000 and Over	100,000-499,999	50,000-99,999	10,000-49,999	2,500-9,999	Under 2,500
All sizes	100.0	24.9	21.1	10.3	21.9	11.5	9.7
500,000 and over	100.0	59.9	9.6	6.3	12.7	5.7	5.5
100,000-499,999	100.0	10.0	58.5	4.5	13.6	7.1	6.2
50,000-99,999	100.0	13.4	13.4	45.7	13.6	6.4	7.3
10,000-49,999	100.0	12.5	14.9	8.0	47.5	9.3	7.7
2,500-9,999	100.0	10.1	15.1	9.0	19.7	34.6	11.3
Under 2,500	100.0	8.8	14.8	9.0	28.1	14.8	27.7

striking findings in this table are that 45.9% of the graduates whose prior residences were in communities of from 2,500 to 10,000 population are practicing in communities of less than 10,000, and that 27.7% of those whose prior residences were in communities of less than 2,500 have returned to communities of this size to practice. These findings coincide with the results of a study of 545 graduates of the University of Minnesota School of Medicine reported at the 1951 Congress on Medical Education and Hospitals by Dean Harold Diehl.

Table 8 shows the percentages of 1935 and 1940 graduates of public and private American medical col-

TABLE 8.—*Percentage of 1935 and 1940 Graduates of American Medical Colleges in Private Practice and Whose Prior Residence Was in Communities of less than 10,000 Population*

Form of Medical College Control	Percentage of Private Practitioners	
	Practicing in Communities Under 10,000	Prior Residence in Communities Under 10,000
Total	20.5	24.3
Public	25.1	31.5
Private	17.8	20.1

leges in private practice who are practicing in communities of less than 10,000 population, and the percentages whose prior residence was in communities of less than 10,000. A definitely larger proportion of graduates of publicly supported colleges are in private practice in communities of less than 10,000 as well as a larger proportion of those whose prior residence was in communities of less than 10,000.

In an effort to evaluate the relative importance of residence before entering medical college and the location

of the medical college in determining the location for practice, table 9 was developed. This tabulation appears to indicate definitely that previous residence is a more potent factor than location of the medical college in determining the ultimate location of the graduates.

Although specific information in regard to the location of internship was not requested in the questionnaires, more than 1,000 graduates provided such information. This offered an opportunity to study for this group the relationship between the place of practice, the location of internship, the college of graduation, and prior residence (table 10). This table is somewhat complicated, and at first sight the columns do not appear to add up to 100%. However, the various combinations of relationships require a duplication of certain figures in each column. If such duplications are eliminated, the accuracy of the figures becomes apparent. This tabulation appears to indicate that prior residence either alone or in combination with location of medical college or place of internship is a more potent influence in determining the place of practice than either of the other two factors.

SUMMARY

Certain data relating to trends in the distribution of physicians have been presented. There are undoubtedly many factors involved in the choice of location for practice by the graduates of our medical colleges. The data here presented appear to indicate that economic factors are of some influence. Although not presented in detail in this preliminary report, the variation in the distribution of the graduates of the individual colleges suggests that the nature of the educational program and the environment in which it is conducted are factors that influence the distribution of the graduates.

Although there is evidence that the location of the medical college attended and the place of internship may be influential factors, it is apparent that residence before admission to medical college is in general the most potent factor in determining the size of the communities in which the graduates of our medical colleges locate.

These findings suggest that an increase in the number of students from smaller communities enrolled by the medical colleges would probably result in an increased number of practitioners for the smaller communities of the country.

766 Irving Ave.

TABLE 9.—*Distribution of 1935 and 1940 Medical College Graduates in Relation to Place of Prior Residence and Location of Medical College, by Relation Between Location of Medical College and Place of Practice*

Relation of Place of Prior Residence and Location of College	Total Number of Graduates	Percentage Distribution of Graduates in Relation to Location of Medical College and Place of Practice: Total	Same City	Other City in Same State or Province	Other State in Same Region	Other Region	Other Country
Total	6,756	100.0	19.3	28.0	13.6	35.6	3.1
Same city	1,804	100.0	48.8	17.4	8.4	22.1	3.1
Other city in same state or province	2,321	100.0	10.4	58.7	7.9	21.5	1.2
Other state in same region	773	100.0	7.2	9.6	62.1	19.3	1.3
Other region	1,637	100.0	6.5	6.5	5.4	80.4	0.9
Other country	122	100.0	2.5	4.1	3.3	7.4	82.0
Not specified	99	100.0	19.2	32.3	10.1	36.4	2.0

TABLE 10.—*Percentage Distribution of 1935 and 1940 Graduates of American Medical Colleges, by Relationship Between Place of Practice and Places of Internship, Medical College, and Prior Residence*

Relationship	City 1935	City 1940	State 1935	State 1940
Total	100.0	100.0	100.0	100.0
Practicing in same place as internship	22.2	22.2	49.6	43.7
Medical college and prior residence also in same place	8.7	9.7	33.4	25.9
Medical college also in same place	4.4	4.1	5.0	3.9
Prior residence also in same place	4.4	3.3	6.8	6.6
Internship alone in same place	4.6	5.0	4.4	7.2
Practicing in same place as medical college	18.5	18.6	49.8	41.8
Internship and prior residence also in same place	8.7	9.7	33.4	25.9
Internship also in same place	4.4	4.1	5.0	3.9
Prior residence also in same place	3.1	3.1	9.7	10.8
Medical college alone in same place	2.3	1.7	1.7	1.2
Practicing in same place as prior residence	29.5	31.9	61.4	54.7
Internship and medical college also in same place	8.7	9.7	33.4	25.9
Internship also in same place	4.4	3.3	6.8	6.6
Medical college also in same place	3.1	3.1	9.7	10.8
Prior residence alone in same place	13.3	15.7	11.6	11.4
Practicing in a different place	59.1	57.3	27.4	32.9

Empirical Approach to the Electrocardiogram.—In the final analysis the electrocardiogram, like other clinical data, is interpreted empirically. It is as good as the judgment of the interpreter.

Any kind of electrocardiogram may be normal in a person under 30 years of age. After the age of 70 almost any change in the electrocardiogram may be merely a part of the aging process. An isolated abnormal electrocardiogram does not inevitably indicate organic heart disease. It may imply some temporarily disturbed function, as in the innervation of hormonal control of the heart, constituting part of the picture of psychosomatic disturbances, and may actually have been engendered by fear or excitement associated with the taking of the record.

Even an abnormal electrocardiogram associated with organic heart disease may be benign or occult, requiring no alteration in the manner of living of the patient. The abnormal electrocardiogram is less important than the circumstances under which it occurs. Taking an electrocardiogram does not absolve the clinician from also using sound clinical judgment. Contrariwise, a normal electrocardiogram is no guarantee that the heart is normal. The electrocardiogram is only part of the clinical examination, an important adjuvant which should never be omitted in a patient with heart disease or in a person in the coronary age group. Under such circumstances it serves as a scanning test, possibly throwing out hints to the alert clinician to examine his patient more carefully.

Experience has shown that the clinical utility of the electrocardiogram depends on the information it gives about several of the physiologic properties of the heart, including its irritability, rhythmicity and conductivity. It furnishes no information concerning the contractile power of the heart or its tone.—Louis N. Katz, M.D., Advances in Electrocardiography, *The Medical Clinics of North America*, January, 1952.

ESTATE PLANNING FOR PHYSICIANS

PART 1: THE BUSINESS SIDE OF THE PHYSICIAN'S LIFE

W. S. McClanahan, Chicago

This is one of a series of articles being developed for physicians who are interested in problems relating to economic security in their business and home activities. Other articles on estate planning and insurance will be published in THE JOURNAL *soon.*—ED.

The physician, like most other men, is a many-sided person. In his practice of medicine he is engaged in a highly skilled calling, is a member of one of the most respected professions, and is usually self-employed. With the great progress in medical education in the past few decades, there is no doubt that the present day medical school graduate has received a satisfactory technical education. Probably no other profession can claim that its members enter practice better equipped by technical training and experience to perform their daily tasks.

In these articles another aspect of the life of the physician that is not directly related to his practice of medicine will be examined. Perhaps it can best be described as "the business side of the physician's life." The scope of the articles may be somewhat boader than that phrase indicates. The physician's life and work as a citizen, businessman, property owner, taxpayer, and head of a family group dependent on him will be surveyed. When the physician's life is analyzed from these viewpoints, it becomes evident that most of his hopes and dreams, his successes and failures, his problems and achievements are common to men in other businesses and professions. One difference seems to be that the physician, by reason of his long and specialized technical training, may not be as well equipped to cope with some of these problems as his counterparts in the fields of commerce and industry.

THE PHYSICIAN AS A BUSINESSMAN

The reasons for this are not difficult to find. The fact that the medical course is now the longest, toughest, and most expensive in the professional field [1] precludes the medical student from engaging in studies in any other field. Perhaps his last taste of business education was a course in commerce, economics, or political science in his liberal arts or premedical course. During his internship and residency he has little time for such studies and almost no daily contact with the business world outside.

Thus the young physician arrives at the threshold of active practice after 8 or 10 years of the best specialized training that our colleges and medical schools can give him. He proceeds to start his own practice and looks forward to his first real earnings. From the viewpoint of these articles, he has "set up his own business" with little previous education or training in the complex world of modern business. Our formal educational system has spent a vast amount of time, effort, and money to make him a good doctor, and very little to make him a good businessman.

Unlike some other professions, such as law and accounting, the practice of medicine brings the physician into very little contact with the business world. The young lawyer and accountant commence at once to handle many types of business transactions for their clients, and thus gain valuable experience that is helpful in the management of their own affairs. The only business experience that the physician gains is in the management of his own property and estate as he acquires it. This learning by trial and error in the fields of business and investments often costs the physician his first few hundred or few thousand dollars of savings, which makes it rather expensive education.

What the physician should recognize, early in his business and professional life, is that modern business is a technical and complex field. It involves a knowledge of economics, finance, banking, insurance, law, and taxes. Many of these fields are as specialized as medicine itself. Judge Waugh in his excellent article [2] stated the case for the legal field well when he said, "In the field of law he (the physician) is no longer a professional, he is a layman." The same can be said of the other fields mentioned above.

Fortunately, for the physician and for other professional men and property owners, there has developed in the past few years a new field of endeavor called "estate planning." Within this field there are now many trained, full time specialists who can assist property owners in planning their affairs to meet today's complex problems. These men are often referred to as the "estate planning team," which generally includes the lawyer, the life insurance underwriter, the trust officer, and the accountant. It is the purpose of these articles to point out how the physician can, with the help of these specialists, solve his financial problems to the benefit of himself and his family.

In a recent issue of THE JOURNAL an article by John Alan Appleman [3] gives a good description of the philosophy of estate planning (he prefers the term "estate analysis"). Appleman sketches a financial picture of a hypothetical case and shows what pitfalls exist for the physician who attempts to be his own financial and investment expert. He also gives an excellent description of the materials and information needed for an estate analysis.

DEFINITION OF ESTATE PLANNING

"Estate planning" is a term that has come into prominence only recently, although the art and practice of estate planning has been carried on for generations.

Member of the Illinois Bar Association and Assistant Vice-President, Lake Shore National Bank.

1. Dickinson, F. G.: Income Tax Discrimination Against The Professions, J. A. M. A. **142**: 1357 (April 29) 1950. Dr. Dickinson estimates that the total cost to the student of the nine year training period is about $35,000, including tuition and school expenses, lost income which he could have earned, and interest on his total investment. In addition the medical school has spent another $10,000 on his education.

2. Waugh, W. F.: Wills for Physicians, J. A. M. A. **144**: 842 (Nov. 4) 1950.

3. Appleman, J. A.: Even Physicians Die, J. A. M. A. **147**: 1547 (Dec. 15) 1951.

There are many misconceptions of the meaning of this term, and it may be well to dispel some of the mystery surrounding it. Perhaps the term itself connotes some narrow limits that do not exist. The word "estate" may indicate to many that this type of planning is done only by the very old and very wealthy, or when death is imminent. Some may think that estate planning is only a device by which the idle rich avoid paying their share of the nation's taxes. Nothing could be further from the truth.

Estate planning has been defined as "the careful and orderly analysis of the family, financial and business affairs of a property owner, while he lives, and the adoption and carrying out of an individual plan for the use of his property and earnings according to his wishes, both during his life and after his death." Perhaps we can simplify it still further. It is the planning of a person so that his property and earnings will do him the most good while he lives and his family the most good after he is gone.

As thus defined, estate planning does not seem mysterious or complicated. A generation or two ago it was not. The usual estate plan in those days, if such it can be called, consisted of a simple will of the head of the family. It was often prepared with little advance consideration, on the occasion of the death of a relative, departure on a long trip, or some other event that brought the uncertainty of life into the property owner's thinking. Such a will usually left the entire estate to the wife, appointed her executor, and excused her from furnishing a surety on her executor's bond. The property owner then signed the will, placed it in a safety deposit box, (or perhaps in some secret filing place in the home, where it might never be found), and relaxed with the satisfaction that comes when a disagreeable task has been completed.

INCREASED NEED FOR ESTATE PLANNING

A generation or so ago that was enough of an estate plan for the average family. Whether we look on those as "the good old days" or not, life was far less complex than it is today. The duties and problems of the business or professional man rested more lightly on his shoulders and he had a better chance to achieve his chosen objectives. By careful management of the financial affairs of the family, he could acquire and hold a substantial amount of property during his working years. Properly invested, this estate would produce sufficient income to support him in old age and his family after his death. The picture today is vastly different. Many changes in the last few years have altered our business and financial lives. We need mention only a few to set the problem out in bold relief.

A national weekly news magazine [4] recently published an interesting study on the effect of rising taxes and declining purchasing power of the dollar. It is stated that the man with a wife and two children who earned $10,000 in 1939 needed $25,000 in 1951 to furnish the same standard of living for his family. In 1939 income tax took only about $350 of the $10,000, leaving $9,650 of disposable income for the family. Due to the decline in purchasing power of the dollar, this family now needs about $18,500 of disposable income to maintain the same standard of living. In 1951 the head of this family would have to earn $25,000 and pay income taxes of $7,500 in order to retain $18,500 of disposable income. On the same basis, the man who earned $15,000 in 1939 would need $43,000 in 1951 to maintain his former position.

These figures point out how very difficult it is today for a salaried or professional man to save enough out of his earnings during working years to build an adequate estate. Even if he is successful in accumulating considerable property, the income alone will not support him at retirement or his family after his death, as it would have just a few years ago. This is the result of a third factor in our current economic life, the decline in interest rates in the past 30 years. Thirty years ago an estate of $100,000 invested in high grade bonds would have produced an income of $5,600 annually, 25 years ago the same estate would have produced $4,700, and today would produce about $2,900. This is the principal reason why the laws of most states have been changed to allow trustees to invest a portion of their trust funds in common stocks, in order to increase the income for the support of the beneficiaries.

These three factors alone (and there are many others) make the financial planning of the average family today more difficult and more necessary than ever before. For professional men, and particularly for physicians, careful and sound financial planning is absolutely essential to a happy and successful life and practice. There are several reasons why physicians need this type of planning more than the average businessman or corporation employee. To check this statement, let's go back to fundamentals. What are the basic reasons that men save money from their day to day earnings? Why not spend at once what is earned each day?

REASONS FOR ACCUMULATING SAVINGS

It has been said that there are only four basic reasons why men save money. First, they save for emergencies such as temporary loss of earnings, a costly illness in the family, or any similar emergency that strikes without warning and requires immediate cash expenditures. Second, they save for disability, so that funds may be available in case the principal income producer of the family becomes unable to work and earn, either for a long period or permanently. Third, they save for their old age, so that they may have some money and source of income when old age makes them unable to continue full time work, or younger and fresher talents crowd them from the stage. Fourth, they save for death, so that their families will have something to fall back on if premature death keeps them from carrying out their other plans.

These four hazards—emergency, disability, old age, and death—are the only reasons why persons save money; and every one of them is beyond personal control. Men cannot, by personal efforts, eliminate the possibility of their happening to them, nor can they choose the date on which they will happen. What they can do is to recognize the risks involved and do everything in their power to plan for them. Then, when the blow falls, the

4. $4,000 man in '39 Needs $9,000 Now, U. S. News 31: 13 (Nov. 16) 1951.

financial effect on them and their dependents will be minimized, if not entirely eliminated. Is the physician more subject to these economic hazards than salaried persons and businessmen? Probably not. If and when one of them strikes, may the financial effect on the physician and his family be greater? Probably yes.

First, as to emergencies. Being self-employed, most physicians do not have the risk of temporary unemployment or being laid off in a slack period that the salaried employee may have. A serious illness or operation in the physician's family may not cost him as much as it would other persons. If he is disabled for even a short period, however, his earnings cease immediately, while the average business employee may receive his regular salary during a disability of several months' duration. If earned income stops, something must replace it. This is a hazard that the physician must provide for through cash reserves, health and accident insurance, and other forms of protection.

The second hazard, disability, is similar to the first, except that it refers more to prolonged illness or incapacity, or permanent disability forcing an early retirement. Its impact on the physician and his family is immediate and may be disastrous. A salaried person may receive considerable help from his employer. If he has several years of service, he may be entitled to a disability pension for life. Here again the physician must do his own planning and protect himself through insurance, savings, and reserves in other forms of property.

In regard to the third hazard, old age, two new elements must be considered. It is not really a contingency, because it is bound to happen to everyone, and the date of its arrival can be calculated with some degree of accuracy. It is here that the true field of financial and estate planning is entered. The economic effect will be greater than that of a temporary emergency, but there is a longer time to plan and act to meet it. It is in this situation also that the physician's greater need for estate planning than that of business employees stands out in bold relief.

SOCIAL SECURITY BENEFITS

There are two forms of retirement income available to most salaried persons in which the physician does not share. The first is Federal Old Age and Survivors Insurance, which now covers about 45 million of the 62 million employed persons, or about three out of four.[5] Under the 1950 amendments to the Social Security Act, about 10 million new persons were brought within its coverage, including over 4 million self-employed persons.[6] Physicians in private practice and other professional men were specifically excluded in these amendments, and there is little possibility of their inclusion in the near future.

There is one exception to this, however, and that is the physician who was in military service between September, 1940, and July, 1947. The 1950 amendments provided that, depending on his total time in military service, the family of a physician who dies within a few years after leaving the service may be entitled to survivors benefits that are valuable and substantial. These benefits might be worth $10,000 or more to the average doctor's family, depending on the age of his dependents at his death. Following is a hypothetical case of a physician who was never employed on a salary and hence never covered under Social Security, except by reason of the special provisions for military service.

This hypothetical physician, Dr. Richard Roe, entered military service Feb. 1, 1942. He was honorably discharged Feb. 1, 1946, and died Feb. 1, 1952. He was survived by his widow, age 45, a son, aged 12, and a daughter, aged 8. His survivors would be entitled to the following benefits:

Benefits to widow and two children until son attains age 18 on Jan. 1, 1958, at $58.50 per month	$ 4,200
Benefits to widow and one child until daughter attains age 18 on Jan. 1, 1962, at $54.80 per month	2,600
No further benefits until widow reaches age 65	
Benefits to widow from age 65 to her death at age 78 at $27.40 per month	4,200
Total benefits received, based on assumed facts	$11,000

The survivors benefits available decrease in amount as the period of time elapsed after leaving military service lengthens, and the right to benefits ceases after several years. It would be well worthwhile for every physician who was in military service to check the nearest office of the Social Security Board and find out what benefits his family would receive in case of his death and when this coverage expires.

For the salaried person who is fully insured under the Social Security Act, the increased benefits under the 1950 amendments are now a considerable factor in planning for retirement. A person who has earned the maximum credit (an average salary of $300 or more monthly), and whose wife is also 65 when he retires, receives retirement benefits for life that may range from $10,000 to $20,000, depending on their longevity. Following is an example of the benefits that might be received by this fully insured employee and his wife after retirement when the husband dies at age 77 and the wife at age 78:

Husband receives $80 per month for 12 years	$11,520
Wife receives $40 per month for 12 years	5,760
Lump sum benefit at husband's death	240
Wife receives $60 per month for 1 more year	720
Total benefits based on assumed facts	$18,240

Since the physician does not have this form of retirement income available through Social Security, he must do his own planning and secure similar benefits through life insurance or annuities. The cost to the physician will depend on whether he purchases insurance or annuities early in life, or over a period of years, with benefits to commence at retirement, or waits until his retirement date and purchases them to commence immediately. In the latter case an annuity to pay him about the same benefits as Social Security would cost about $20,000.[7]

5. Annual Statistical Supplement, Social Security Bulletin, vol. 14, no. 9, Federal Security Agency, (Sept.) 1951.

6. Your Social Security Benefits, Industrial Relations Institute of Commodity Research Bureau, Inc., New York, 1951, p. 4.

7. This is not an exact comparison, as the insurance companies do not issue annuities that will pay the annuitant in exactly the same amounts and methods as Federal Old Age and Survivors Insurance. If the insurance or annuities are purchased earlier, with benefits deferred until retirement, the costs will be substantially less. The cost of this and other annuities mentioned in this article were taken from standard 1951 insurance handbooks and represent an average cost based on the rates of several companies, rather than the quoted rate of any particular company.

PRIVATE PENSION PLANS

The second form of retirement income available to a large number of salaried persons employed in business and industry is the private pension plan. If the plan is approved by the Bureau of Internal Revenue, the employer's contributions are deductible from his income tax return and are not taxable to the employee until he receives the funds at retirement. Thousands of these plans have been adopted recently, and no figures are available as to how many persons are covered by them or the average pension payable.[8] Let us assume that a company has a private pension plan that will pay a salaried employee $200 per month for life after he retires at 65. If the physician wishes to purchase similar retirement benefits in the form of life insurance or annuities, he will find that the costs are substantial. An annuity purchased at age 65, with payments of $200 per month for life to commence immediately, with no refund in case of early death (which resembles the provisions of many company pensions), would cost around $30,000. If the funds are built up on the books of an insurance company by the purchase of life insurance or annuities over a period of years, the compound interest principle will substantially reduce the cost of this retirement income.

These are two forms of retirement income that are an important part of the estate planning of most businessmen and salaried persons, and to which the employer makes a contribution. No employer is making a contribution to any retirement fund for the practicing physician. Neither can the physician lay aside from his earnings similar amounts tax free, and achieve the income tax deferment advantages of the private pension plan. These are two additional reasons why the physician is more in need of sound financial planning than many other persons if he is to enjoy the same benefits at retirement.

TAX-FREE RETIREMENT FUND FOR PROFESSIONAL MEN

At present there is some indication that this so-called income tax discrimination against the professional man may be abolished, or at least reduced substantially. The physician is not alone in this inability to join in a pension plan and accumulate tax free funds for retirement. Lawyers, accountants, and other self-employed professional men are also denied this privilege. In recent years the pressure of high income tax rates has caused much agitation among professional groups for legislation to end this tax discrimination. A special committee of the American Bar Association has been actively at work devising plans to amend the income tax laws in furtherance of this objective. Their work culminated in a bill introduced in Congress on June 7, 1951, known as the "Keogh Bill" or H. R. 4371, which would have granted to professional men the privilege of contributing a limited portion of their earnings, tax free, towards a "restricted retirement fund." [9] The general principle involved in this bill has the support of many professional groups, including the American Medical Association.[10] The chairman of the special committee of the American Bar Association has stated that this bill, or one similar to it, has a fair chance of being enacted into law at the next session of Congress, if it has the active support of all professional groups.[11] Such legislation would have a most beneficial and far reaching effect on estate planning for physicians.

The fourth and last of the reasons why people save is to provide for their dependents in case of premature death. Everyone likes to think that, if he is allowed to live to carry out his plans, he will be able to provide for himself and his dependents through his own efforts. But the mortality tables contain the undeniable proof that all men will not be granted this privilege. No financial plan is sound that does not give this hazard a high priority in planning and allocate sufficient proportions of the available resources to insure against it. The worst tragedy that can happen to any family unit is the premature death of its principal income producer. The death of any other family member is a major tragedy, and results in great emotional shock and sense of loss to the remaining members, but as an economic unit the family may not be seriously affected. When the head of a family, the "breadwinner," is taken prematurely by death, the very existence of that family as a unit is seriously threatened. Only when the head of the family has worked out a sound financial and estate plan, and has implemented it with adequate measures to assure its success, can his family be reasonably sure of their ability to stay together as a family, not dependent on relatives or on charity. This sense of family responsibility and loyalty is the underlying reason for all financial and estate planning. Later articles in this series will point out some of the methods by which these purposes and objectives can be achieved.

605 N. Michigan Ave.

8. The latest official figures of the Treasury Department released in August, 1946, estimated that there were in existence about 6,800 pension plans covering more than nine million employees. Unofficial estimates by a national tax reporting firm estimated that there were 13,000 pension plans and 3,000 profit sharing plans in effect as of July, 1951.

9. Identical bills were introduced in the House by Representative Keogh (H.R. 4371) and Representative Reed (H.R. 4373) and referred to the Committee on Ways and Means. On July 25, 1951, Senator Ives introduced in the Senate an amendment to the Revenue Bill (H.R. 4473) then pending, which was in substance similar to the other bills. The Senate Finance Committee voted to take no action on this amendment, but referred it to its technical staff for study. Report of The Special Committee on Retirement Benefits, distributed to members of the American Bar Association, Chicago, Sept. 17, 1951.

10. Dickinson.[1]

11. Committee Report, American Bar Association.[9]

Nature's Sugar Tolerance Test.—Examination of the urine for sugar is particularly valuable in the febrile patient. Infection is nature's sugar tolerance test; in many instances fever taxes the function of insulin far more than 100 gm. of glucose. The alert physician will, therefore, use these occasions to make his routine urinalysis for sugar. If negative, the patient probably has a good insulin reserve. If positive, one must suspect and usually treat as diabetes, establishing the severity of the disease later by appropriate study.

Part of the mechanism which makes fever antagonistic to insulin is the activation of the adrenal cortex. The proper examination of the urine for sugar during the treatment with ACTH and cortisone may well reveal a number of prediabetics. Endocrine diseases, such as hyperthyroidism, acromegaly, and Cushing's syndrome, have a high incidence of diabetes. Repeated examinations for diabetes are part of the management of such patients.—F. D. W. Lukens, M.D., The Proper Use of Laboratory Tests in the Diagnosis and Management of Diabetes, *Diabetes*, January-February, 1952.

REHABILITATION OF PATIENTS WITH DEEP BURNS

James Barrett Brown, M.D., St. Louis

Patients with large full thickness loss of skin from burns require skin grafting for healing, function, and rehabilitation. This group presents a major part of the burn problem, and a summary of their needs and their care is outlined here.

In the extensive literature, much effort has been directed toward some method of local care, to taking care of acute and chronic shock, and to problems of deranged blood chemistry and metabolism. This work has been of great benefit and has stimulated a burn consciousness, so that excellent care may be afforded burned patients, and many more lives are saved, thus adding to the numbers who require later grafting and reconstruction. There still is, however, a lack of reports of completed care of patients who have been badly burned and not only have been saved from death but have had satisfactory surgical repair of open wounds and contractures and have been rehabilitated to useful life.

Fig. 1.—Deep widespread burn (Air Force) requiring resurfacing of nearly the whole face, reconstruction of all four eyelids, the nose, both ears, and both eyebrows, and grafting of both hands. (The patient was cared for at Valley Forge and St. Louis Veterans Hospitals and later attended college and was married.)

One difficulty arises from interpretation of "superficial" and "deep" burns and the healing processes of both. The recording of various degrees of burns may be a stumbling block, and the degrees could be replaced with a classification of "superficial," "partial thickness," and "full thickness" loss, with "deep burn" reserved for full thickness loss. Superficial burns may not have any deep areas, but deep burns nearly always have superficial areas. The determination of these depths is often difficult and should lead to a conservative attitude toward immediate or early excision of burns.

If full thickness of skin is burned away over a large area, there will be contraction and distortion and loss of function unless the lost skin is restored. Epithelium will regenerate, but the full complement of the skin will not. The patient will require a pad of derma to receive the blows and trauma and to adjust to the temperature changes of his environment.

A basic plan of repair of large full thickness losses of skin may be (1) to get the area as clean as possible, as soon as possible, and (2) to restore what skin has been lost. This should reduce scar formation to a minimum; then, when the wound is healed and the patient has recovered, one may proceed with any necessary secondary repairs for the release of unavoidable distortion or for restoration of features and contour anatomy and function.

HEALING OF BURNS

Almost any treatment that promises as little work as possible for the fresh burn is apt to become popular; however, so far no application has been found that will produce normal skin in wide full thickness losses. When it is claimed that a certain preparation or type of treatment will insure complete healing without scarring, it apparently is not generally realized how healing occurs in either superficial losses of skin or in full thickness losses of skin.

If there is rapid healing and return to function following a burn without much scarring or deformity, this means that the full thickness of the skin has not been lost and that regeneration has occurred from the deep glands in the derma, much the same as occurs in the donor site of

Fig. 2.—Burn (Air Force) with loss of vision from scarring of lids, requiring reconstruction of all four eyelids, both brows, all four canthal regions, and the right ear. The patient also had complete grafting of both hands, palmar and dorsal surfaces. (The patient was cared for at Valley Forge and St. Louis Veterans Hospitals.) Restoration of sight, function, and normal anatomic contour. The patient later graduated in law.

a thick split graft. It has been observed that healing of these struggling cells is interrupted by chemical, bacterial, or mechanical trauma. Therefore, it appears that cleanliness, protection, and the avoidance of strong chemicals will promote this type of healing, and these objects of treatment have stood the test of time. It is obvious that a dirty dressing, leaving a coating of pus over a partial thickness burn, would be apt to stop healing and that such a partial loss might be changed to a full thickness loss. This same finding is true in coagulum methods of treatment, whether the coagulum is formed by chemicals or is a natural one on the wound. It seems unnecessary to mention that tannic acid is the chemical used to turn hides into leather and that such strong chemicals can be injurious to healing tissue.

From Department of Surgery, Washington University Medical School.

Some of the illustrations have been omitted from THE JOURNAL but will appear in the author's reprints.

Read before the Section on Surgery, General and Abdominal at the One-Hundredth Annual Session of the American Medical Association, Atlantic City, June 13, 1951.

PRIMARY CARE

With the chance to see burns early, to clean them if necessary, and to get a comfortable fine mesh grease gauze, pressure-fixation dressing on them, it does not seem necessary to leave the burns exposed without dressings and require all patients to be hospitalized. For

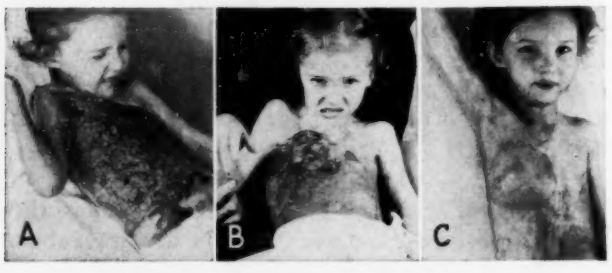


Fig. 3.—*A*, deep burn of the axilla, arm, and neck. *B*, flat surface grafting with healing and distorted position. *C*, secondary restoration of function with free thick split grafts for full axillary coverage and neck reconstruction.

superficial burns (for which the exposure method is best adapted) almost any careful treatment will suffice, if deep loss of derma can be avoided. Many of these burns will heal in 10 to 12 days with protective dressings, and many of them do not require hospitalization. In times of disaster this seems most important, and patients in hospitals for exposure treatment might actually give aid to others if their own burns were dressed and did not require hospitalization. There have not been sufficient observations to permit a final decision that the exposure method is ready for complete adoption for large deep burns. For burns in some areas, such as the anal and genital regions, in some facial burns, and in some instances in children, where hospitalization is necessary, this method may find good usage. The decision in mass burns or disaster would seem to be one based on proper consideration of the patients in relation to personnel, supplies, and available beds as well as what might be best for the patients. Using the best care in individual instances is proper, but it may be necessary to drop down to secondary levels as pressure of work and availability of materials dictate.

One important consideration is that the care of deeply burned patients is complicated and time consuming. The first echelon of care may have to center on the easiest

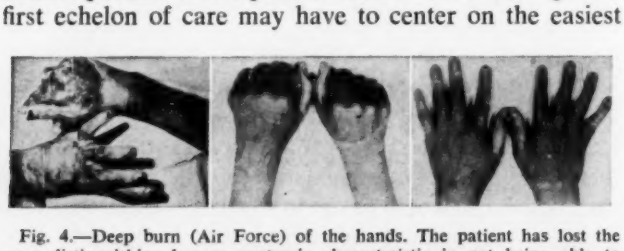


Fig. 4.—Deep burn (Air Force) of the hands. The patient has lost the most distinguishing human anatomic characteristic in not being able to bring the thumb around in front of the fingers. Rehabilitation was accomplished with split graft resurfacing of both hands at the same time. (The patient was cared for by two surgical teams at Valley Forge Hospital.)

plan, but this may necessitate greater total care as the problem of the deep burn looms in the rehabilitation. There are many ways of looking after a burn primarily; the paraffin treatment for example is one of the best and kindest methods devised. Colebrook's germ-free air is ideal in his hands with his equipment. But all of these methods cannot always be carried out, so that, in deciding on an over-all treatment, we do not have to find argument over no dressings or pressure dressing, but we may be sure to employ careful and gentle methods and to avoid methods that have real pitfalls. Tannic acid had its great advocacy because of the apparent ease of care, and it, silver nitrate, and other irreversible methods are things to avoid. Such procedures as amputating arms because the hands are burned (as was carried out by the enemy in World War II) form the most irreversible procedures. Skeletal traction through finger tips does not appear warranted routinely to elevate arms, and in a large series would probably do as much harm as good.

Amputation may be considered at times for hopelessly damaged lower extremities in an effort to control the fatal tendencies, and this has been carried out successfully by Dr. Carl Moyer. Severe deep burns of the lower extremities and the body in general cause death after the immediate shock has been controlled. Often, despite an inadequate blood chemistry explanation, a possible cause of death may be the destruction by the burn of the nocifensor system, Sir Thomas Lewis' name for a system of

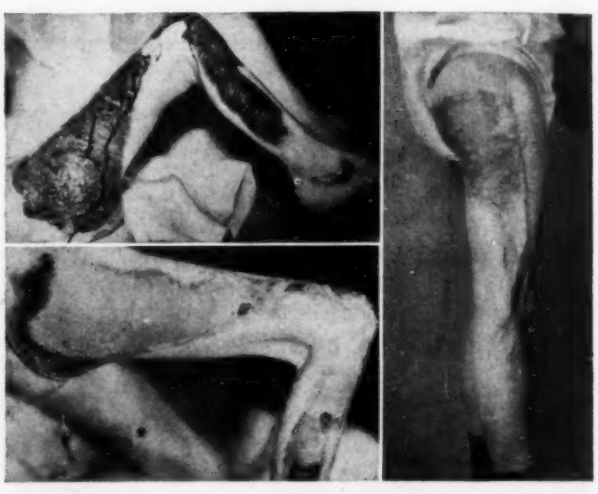


Fig. 5.—Homografts from the patient's father were used as a lifesaving measure. There was a deep burn with such severe pain reaction that recovery was questionable. The patient "walked around" in homografts; later final rehabilitation was accomplished with his own skin as shown. (Brown, J. B., and McDowell, F.: Skin Grafting, ed. 2, Philadelphia, J. B. Lippincott Company, 1949.)

nerves in the skin and mucous membranes, which are concerned with local defense against injury.

In allowing nature the best chance of healing, protection of the wound with fine (no. 44) mesh grease gauze is applicable. Pressure fixation of the burn over this gauze is obtained with large pads, cotton surgical waste, gauze rolls, and bandaging. "Pressure-fixation" is used, instead of "pressure," to indicate support of adjacent joints to put the area at rest. The use of hydroscopic gels, reported by Curtis and being developed by Oakes and Trimbly, may offer an efficacious method.

EARLY GRAFTING

Early grafting is done in deep burns, before irreversible chronic shock and general debilitation occur. Most burns can be ready for grafting in two weeks, and agents and enzymes to hasten the separation of sloughs will prove of great worth. If exposed bone or tendon persists, grafting can be carried up to these areas and as much healing as possible obtained. Flat surfaces can be repaired by excision and grafting, but not the face, genitalia, or feet.

The technique of immediate excision and grafting has not been used in a series of sufficient size so that results could support claims for it. Its use is limited to small areas that have been burned with hot metal or other agents, leaving a definite outline of damage. Certainly this process should not be applied to the face, hand, foot, or genitalia. Even on flat surfaces the results are not promising enough so that it can be advocated as a general procedure, as there is no way of being sure about partial and full thickness losses.

PREPARATION FOR GRAFTING

A fundamental of dressing all raw wounds is that fine (no. 44) mesh gauze be placed next to the surface, so that granulations will not grow up through the coarse meshes of regular surgical gauze, stick, and cause pain. These dressings can be moist with mild antiseptic or saline solution; and when cellulitis is controlled, grease dressings on fine gauze or cotton bed-sheeting can be used. Needless to say, all these dressings on raw surfaces should be kept clean and changed as necessary, so that secretions are not dammed over the surface. A firm pressure or elastic dressing may be of advantage in older wounds, and discolored, edematous granulations may be flattened out into a firm, bright red surface without the use of chemicals or without their having to be cut away.

INFECTION AND CONTAMINATION

Very large open burns, in spite of what one might hope for, probably cannot be gotten or kept bacteriologically clean. They might be made free of infection, but over any long period they are apt to have organisms in them. The essential thing is whether there is infection with cellulitis or just contamination, with the patient possibly living in symbiosis with the organisms. Careful evaluation of the general condition and local area usually suffices for determination of the time for operation.

THICK SPLIT SKIN GRAFTS AND HOMOGRAPHS

For making extensive repairs, such as 250 sq. in. being transferred at one time, it is essential that a method of grafting be used in which prompt healing of the donor site can be had. For this the thick split skin graft is used, with careful cutting of the grafts. If they are cut too deep, added crippling may occur; in some patients, trouble in the donor site has exceeded that of the original lesion.

Homografts are used as emergency "dressings" of wounds to tide patients over critical periods; many lives can be saved by these grafts. There is respite from pain and dressings and fluid loss while the grafts are in place, and the patient may actually walk around in the skin of another person. As far as we know homografts in warm-blooded animals do not survive permanently. Dr. Leo Loeb, one of the foremost geneticists, believes that except for identical twins no two human beings are exactly alike. Successful homografts in identical twins with permanent survival have been reported, and have been studied by Dr. Loeb. If an identical twin is burned, his twin's skin can be used, with good hope of permanent survival.

If corticotropin (ACTH) could cause foreign skin to survive permanently, one of the greatest obstacles would thereby be surmounted. It is difficult to see, however, how the use of corticotropin could make everyone alike, which seems necessary for homografts to survive. There seems to be confusion in the use and terminology of homografts, there seldom being differentiation between "take" and "permanent survival," but so far no substantial series of permanently surviving homografts has been observed. Homografts will take and grow with or without hormones or antihistamines, but permanent survival is yet to be proved. A recent report has been made of growth of homografts attributable to the use of corticotropins, but the grafts reported had not been observed over a long period, and only a single instance was cited. There have been known survivals of homografts for several months without the use of corticotropin. No measurable effect of corticotropin on the permanence of survival of homografts clinically or experimentally has been reported. This does not mean that efforts at producing such a fortunate situation are not laudatory or that they should be given up.

REPAIR OF CONTRACTURES AND REHABILITATION

Technical procedures and short-cuts in early care are of importance, but not so much so as to cloud the fact that full thickness of the skin has been lost in many patients and that a great problem in rehabilitation is the late grafting and repair of contractures and distortions and replacement of lost features. This echelon of plastic surgery is sometimes overlooked. Loss of features and sight, because of damage to the lids, may follow facial burns, and in World War II about one-third of the work in the plastic surgery centers was in repair of burned faces. Even with early grafting of raw areas, lost features still have to be rebuilt.

MILITARY PLASTIC SURGERY

In any area of gasoline usage, there may be many patients with deep burns. The same depth results whenever there is sufficient heat long enough to destroy the full thickness of the skin. Even flash burns may be hot enough to destroy the full thickness on the face and hands, though clothing may protect the rest of the body.

Burned hands are a close second to burned faces in problems of reconstruction, though they respond better to early grafting than do faces. Time-consuming restorations in late contractures make the plan "to get the area as clean as possible as soon as possible and restore what skin has been lost" all the more important.

In preparation for disaster and military surgery, training of young surgeons for flat surface and early hand grafting is important. Army surgeons and foreign observers (and several surgeons on the rotating surgical service of the Washington University Medical School each year) are trained to do good flat surface grafting. In World War II a large part of the work depended on these men, and their good work justified this type of surgical training.

CONCLUSION

The final criterion of the result of reconstruction and rehabilitation is the extent to which the patient is able to resume his normal station in life and carry out his usual activities without embarrassment in regard to his appearance or limitation in his motions. This optimum goal seems to be a high one, but it can be attained surprisingly often by sufficient persistence on the part of the surgeon and others caring for the patient, along with the cooperation of the patient himself.

400 Metropolitan Bldg., Grand Ave. and Olive St. (3)

REHABILITATION OF HAND FUNCTION IN RHEUMATOID ARTHRITIS

Donald L. Rose, M.D., Kansas City, Kan.
and
H. Worley Kendell, M.D., Chicago

Although recent clinical literature abounds with publications that attest to the high level of medical interest in the treatment of rheumatoid arthritis, few [1] of these have dealt with the hand deformities produced by this disease. Yet, the inability to utilize the hands in some useful, or at least time-consuming, occupation constitutes for most patients a more distressing complication by far than similar involvement of any other combination of joints.

There is general agreement as to the importance of the prevention of deformities, and recent publications [2] have shown in excellent fashion the means by which such deformities, including those of the hands, may be minimized. There does and will remain, however, a certain percentage of patients whose hand disability will increase despite attention to these prophylactic measures. We present this report in the hope of interesting others in deviating from the frequent attitude of acceptance of the crippled hand as one of the inevitable evils of rheumatoid arthritis.

ORIGIN OF THE HAND DEFORMITIES

With few exceptions [3] there apparently has been little effort to clarify the mechanisms by which hand deformities occur. Most workers have merely described the deformity as it is seen. Although Comroe [4] defines the hand deformity of rheumatoid arthritis as ulnar deviation of the fingers, hyperextension at the metacarpophalangeal joints and acute flexion of the interphalangeal joints, these are the earlier changes. Later, as shown in figures 3, 4, and 5, with the exception of ulnar deviation of the phalanges at the metacarpophalangeal joints, which remains constant, the reverse of these changes occurs, with flexion at the metacarpophalangeal joints and contracture of the palmar fascia, hyperextension of the proximal interphalangeal joints, and flexion of the distal interphalangeal joints. There are, in addition, subluxation of the proximal phalanges anterior to the head of the metacarpals and loss of space at the proximal interphalangeal joints. Careful questioning of the patient usually elicits the sequence of appearance of these deformities as follows: flexion at the metacarpophalangeal joints (especially the second and third), ulnar deviation of the phalanges at the metacarpophalangeal joints, and, as a later manifestation, hyperextension at the proximal and flexion at the distal interphalangeal joints.

The constancy of these deformities strongly suggests a uniform mechanism for their production common to all persons with this disease. The obvious atrophy of the intrinsic musculature of the hand has given rise to the common belief that weakness of these small muscles accounts for the deformities. However, carefully performed manual muscle tests [5] reveal widespread weakness of all muscles associated with hand function, the intrinsic muscles being no weaker proportionately than the others. In fact, although it is impossible to grade muscle strength accurately because of the pain induced by the test procedure, the lumbricales seem to be somewhat stronger relatively than the other hand musculature.

Attention has previously been directed [6] to the theoretical importance of the dorsal interossei in maintaining the normal position of the phalanges at the metacarpophalangeal joints. The finger flexors and extensors, the lumbricales, and the palmar interossei are fusiform muscles; as such, the muscle fibers transmit their tension directly in the line of movement of the tendon of insertion. The dorsal interossei are bipennate muscles, the fibers of which insert into a central tendon. The efficiency of pull of these fibers varies with the sine of the angle of insertion into the central tendon. The more nearly the fibers insert at a right angle to the central tendon, the less effective is the mechanical force of the muscle contraction; the more acute the angle of fiber insertion, the greater its mechanical effectiveness.

In the patient with rheumatoid arthritis, widespread muscle weakness has already been noted. Continued holding of the fingers in the normal position of slight ulnar deviation of the index finger tends to place the radial half of each of the dorsal interossei on stretch. This additional fatigue and weakness further diminishes the effective pull of these fibers; at the same time the ulnar half of the fiber group shortens adaptively, but at a less acute angle of insertion and with less effective pull on the central tendon. The antagonistic palmar interossei are placed at no such mechanical disadvantage, since they are fusiform muscles. It is believed that the imbalance thus produced between the dorsal and palmar interossei accounts for the component of ulnar deviation in the deformity syndrome.

The component of flexion at the metacarpophalangeal joints and extension of the interphalangeal joints may

---

Read before the Section on Physical Medicine and Rehabilitation at the One-Hundredth Annual Session of the American Medical Association, Atlantic City, June 14, 1951.

From the Department of Physical Medicine, University of Kansas School of Medicine (Dr. Rose) and the Department of Physical Medicine and Rehabilitation, University of Illinois School of Medicine (Dr. Kendell).

1. Joplin, R. J., and Baer, G. J.: Treatment of Arthritic Joints, J. A. M. A. **118**: 937 (March 21) 1942. Law, W. A.: Surgery in the Treatment of Rheumatoid Arthritis and Ankylosing Spondylitis, Proc. Roy. Soc. Med. **41**: 251 (April) 1948. Bunnell, S., and Howard, L. D., Jr.: Additional Elastic Hand Splints, J. Bone & Joint Surg. **32-A**: 226 (Jan.) 1950. Kestler, O. C.: Reconstruction of the Deformed Arthritic Hand, Ann. Surg. **131**: 218 (Feb.) 1950. De Rosa, A. J.: An Exercise Glove, Phys. Therapy Rev. **30**: 138 (April) 1950.

2. (*a*) Hill, D. F., and Holbrook, W. P.: Prevention and Treatment of Deformities in Rheumatoid Arthritis, J. A. M. A. **142**: 718 (March 11) 1950. (*b*) Holbrook, W. P., and Hill, D. F.: Manual of Rheumatic Diseases, Chicago, The Year Book Publishers, Inc., 1950. (*c*) Primer on the Rheumatic Diseases, Chicago, A. M. A., 1949.

3. (*a*) Small, J. C.: The Mechanics of Deformities of the Hands in Atrophic Arthritis and a Discussion of Their Prevention and Correction, Ann. Int. Med. **32**: 1087 (June) 1950. (*b*) Pruce, A. M.: Rehabilitation of the Hand, J. M. A. Georgia **38**: 49 (Feb.) 1949.

4. Comroe, B. I.: Arthritis and Allied Conditions, Philadelphia, Lea & Febiger, 1944, p. 225.

5. Kendall, H. O., and Kendall, F. P.: Muscles: Testing and Function, Baltimore, Williams & Wilkins Company, 1949.

6. Rose, D. L., and Wallace, L. I.: A Remedial Occupational Therapy Program for the Residuals of Rheumatoid Arthritis of the Hands, Am. J. Phys. Med. **31**: 5 (Feb.) 1952.

arise through excessive use of the lumbricales. The characteristic involvement of the proximal interphalangeal joints in this disease gives rise to efforts on the part of the patient to spare himself as much pain as possible in the course of normal flexor use of the fingers. Since the metacarpophalangeal joints are usually not so painful on use, flexion here with splinting of the interphalangeal joints provides flexor function of the hand without the pain of normal use of the part. Flexion at the metacarpophalangeal joints and extension of the phalanges are functions of the lumbricales rather exclusively, and, when employed consistently in this fashion, little use is made of the normal flexor-extensor mechanisms of the fingers.

MATERIAL AND METHODS

Rehabilitation of hand function must proceed along the lines of correcting the muscle imbalances described. However, in formulating a rehabilitation program, we realized early that several provisions must be met adequately:

1. The patient must be well oriented to the general details of the program and his cooperation pledged on a long-range basis. Failure to accomplish this before treatment is instituted may result in loss of the patient after the first enthusiasm of "trying something new" has died away.

2. Clinic physical therapy and occupational therapy, except for the hospitalized patient, are not practical except in the sense of providing supervision of the treatment program.

3. A home program of physical and occupational therapy must be easy and convenient to perform, yet effective. Realistically, it must be admitted that a long system of exercises, complex, expensive, or awkward apparatus, or procedures that require assistance by another person will yield disappointing results, because the patient will not or cannot carry out the procedures often enough to be effective.

4. Any assistive device must be easy to apply or to remove, be acceptable to the patient, be relatively inexpensive, and be correctly designed, not merely to support the part but to assist in the normal functional use of the part.

5. The four "M's" of treatment must be effectively combined: motivation, mobilization, measurement, and medication.

The methods to be described have been employed on nine patients, eight women and one man, whose ages ranged from 43 to 67 years, who by all the usual criteria [2b,e] undeniably had rheumatoid arthritis, and who had had the disease 4 to 27 years. No effort is made to appraise these methods critically, since such appraisal of these or any other therapeutic agents in this disease requires several years of observation. The only element of selection was to exclude patients whose long-term cooperation was doubtful or who did not exhibit the hand deformity that the various illustrations demonstrate.

Correction of the deformity of metacarpophalangeal flexion may be attempted by stretching of the palmar fascia and the anterior part of the capsule of the metacarpophalangeal joints. Finger painting, or the home equivalent with the use of soap suds or glycerin on a smooth surface, after the fashion shown in figure 1 is surprisingly effective in lessening the flexion deformity. In utilizing this procedure, the patient makes a positive effort to touch the palmar surface of the metacarpophalangeal joints to the smooth surface as the fingers are extended. The opposite hand, or the hand of another person, adds counter pressure over the dorsum of the hand and guides the motion of finger extension. The hand prints shown in figure 2 provide a means of motivation as well as measurement of palmar extensibility. Of greater importance, however, is the additional functional use made possible by the increase in extensibility of the fingers.

Correction of the deformity of finger extension can, in the absence of bony ankylosis of the proximal interphalangeal joints, be accomplished by one or more modifications of the use of the inclined plane as shown in figure 3. The traditional instruction to "squeeze a rubber ball" as a means of increasing finger flexion is, in our

Fig. 1.—Extension at metacarpophalangeal joints. *A*, unassisted. *B*, assisted stretching.

opinion, completely futile. Its continued reiteration seems to be one of those rare instances of serial confusion in medical thinking, owing probably to the fact that no one has challenged its use. The human hand functionally is not designed to grasp a spherical object; it is best adapted to the grasp of cylindrical objects. The purpose of the exercise is to increase range of motion in the direction of finger flexion. The resistance of the rubber ball increases with its compression and exceeds the muscle strength or compressing force precisely at the point where additional range of motion is desired. We have employed the inclined plane for two reasons: it is a semicylindrical object well adapted to the function of grasp and the opposing force of the handle surface that resists compression is perpendicular to its surface, thus tending to force the fingers radialward as they grip the handle. Assisted flexion is provided by the other hand, or the hand

of another person, as shown in figure 3*B*. Although this figure shows the inclined plane principle adapted to a sanding block, it is equally adaptable to any handle that must be gripped: a broom, iron, plane, saw, or hammer. The patient is carefully instructed in this procedure, as hasty, overforceful grasp or improper relationship of the remainder of the extremity to the gripping mechanism may traumatize the irritated joints and lead to more post-exercise reaction than necessary.

Correction of the deformity of ulnar deviation at the metacarpophalangeal joints may be accomplished in two ways: through exercise of the dorsal interossei with finger painting activities or by use of a simple assistive device that minimizes the effect of weakness of the dorsal interossei and permits normal flexion and extension of the fingers. Figure 4 illustrates, with the left hand, the widespread abduction of the fingers, emphasizing particularly the effort at radial deviation of the second and third fingers; the assistance of another hand provides guidance for the movement and encourages more finger abduction than initially would be possible otherwise. The paint, which, of course, is freely water-soluble, provides a crude but objective measurement of the degree of finger abduction.

In an effort to provide a continuous measure of support for the fingers, yet not inhibit hand function, numerous splints, casts, and braces of various types were em-

Fig. 2.—Hand prints (right hand with pressure) at weekly intervals following stretching procedure shown in figure 1.

ployed briefly and then discarded, because they failed to satisfy the necessary criteria outlined previously. Small [3a] has described an assistive device for exercise of the hand, but the device illustrated in figure 5*B* differs from it in several important respects. It consists of a simple cotton jersey glove to which are attached hooks and eyes in such a fashion that, when rubber bands are attached to the hooks, the normal action of the finger extensors and dorsal interossei is imitated. The "origin" of these elastic bands is at the wrist, the "finger extensors" are "inserted" on the glove at the dorsum of the proximal interphalangeal joints, and the "dorsal interossei" are "inserted" on

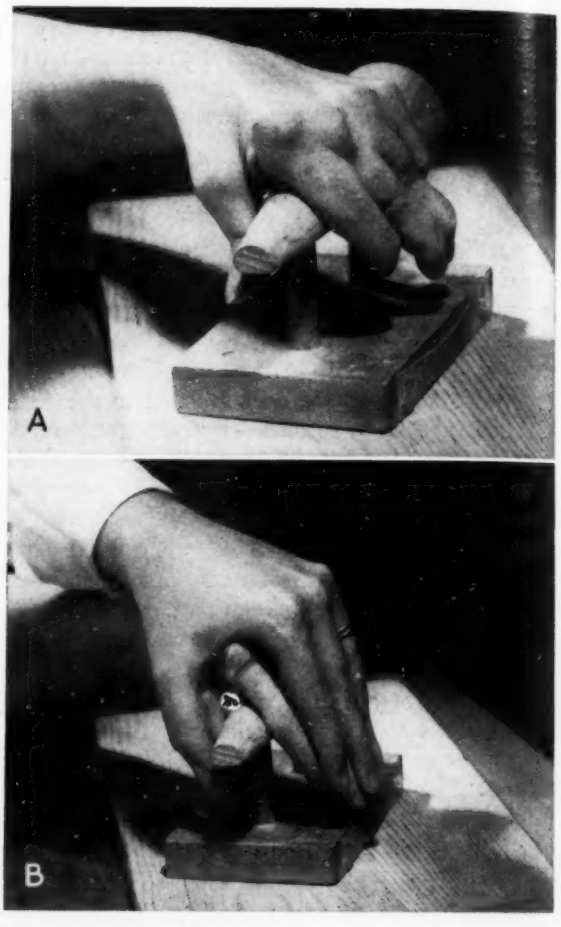


Fig. 3.—*A*, flexion at interphalangeal joints, employing inclined plane. *B*, assisted flexion of fingers in radial deviation.

the glove on the radial side of the distal interphalangeal joints. The eyes provide a means of guiding the elastic bands. By these means, the elastic forces can be varied not only in direction but in intensity as well. The efficiency of this device may be seen in figure 6, where x-ray examination reveals not only complete correction of the ulnar deviation of the fingers but also significant alteration in the subluxation at the metacarpophalangeal joints.

It should be emphasized that this device does not correct deformity directly; it merely holds the part in such a position that hand function can be carried out in a more nearly normal fashion without the continuing trauma of attempted function on a basis of deformity. Although assistance may be given the finger flexors by a similar arrangement of hooks and eyes on the palmar surface of the glove, these are usually unnecessary. The normal functional activity of the hand is one of finger flexion, and the exercise against the resistance of the elastic bands soon produces increased strength in the finger flexors. Further, the presence of hooks and eyes on the flexor surface of the glove tends to interfere some with hand

function. Since the objective of the device is to assist function, anything that detracts from this aim is to be avoided if possible. As with any use of the hand, excessive use of this assistive device will produce increase in joint and periarticular discomfort. It is not necessary to remove the device for rest of the part; merely unhooking the elastic bands at the wrist releases the corrective tension and provides rest. The presence of the glove seems to exert a protective action on the effects of exposure of the part to environmental influences, and patients have commented on the increased feeling of warmth in the hand and decrease in stiffness of the fingers with its use.

Pure exercises have been simplified and purposely held to a minimum. Figure 7 demonstrates, with the hand of a normal person, the basic exercises that have been adapted from Watkins [7] and Nesbitt.[8] Copies of this photograph, rather than written instructions, are provided each patient. It is our observation that it is easier for the patient to attempt to duplicate a modeled action than to follow instructions that state exactly the same thing.

ADDITIONAL MEASURES

Although we believe the procedures described are directed primarily against the deformities produced in the hand by rheumatoid arthritis, it is realized that full advantage must also be taken when possible of other measures if the patient is to derive the maximum benefit from the treatment program. We have employed three such measures:

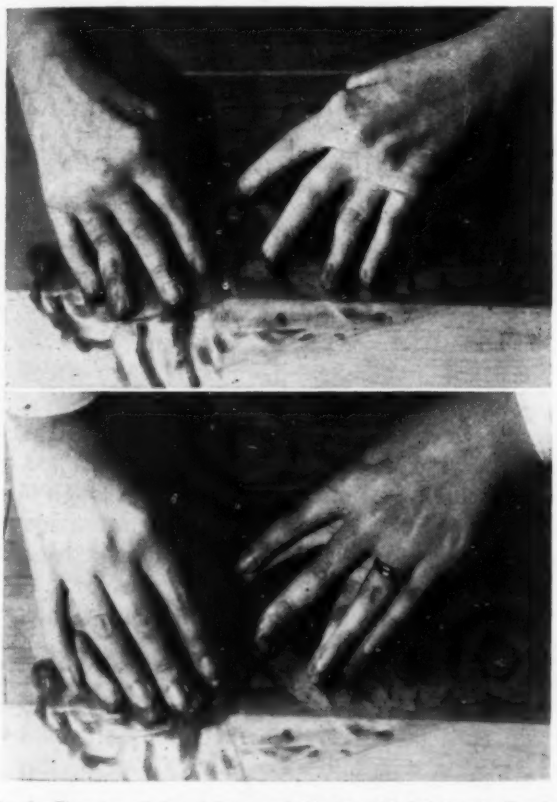


Fig. 4.—Exercise of dorsal interossei (shown with left hand). Emphasis on radial deviation of second and third fingers.

1. *Corticotropin (ACTH) or Cortisone Therapy.*—The action of either corticotropin or cortisone in diminishing rest pain, muscular and articular stiffness, or joint pain and tenderness on motion is well known. Of equal importance from the rehabilitation standpoint is their action in blocking or minimizing increased soreness, stiffness, and swelling of the part following exercise. Obviously, if such reactions can be minimized, the rate of recovery of function will be materially accelerated. On this basis, we have employed these compounds in standard doses for brief, two to four-day periods, particularly at times when the exercise program is to be intensified. When we have employed them in this fashion, we have seen little of the usual flare-up of symptoms on cessation of therapy frequently observed following treatment with these agents over a longer period of time.

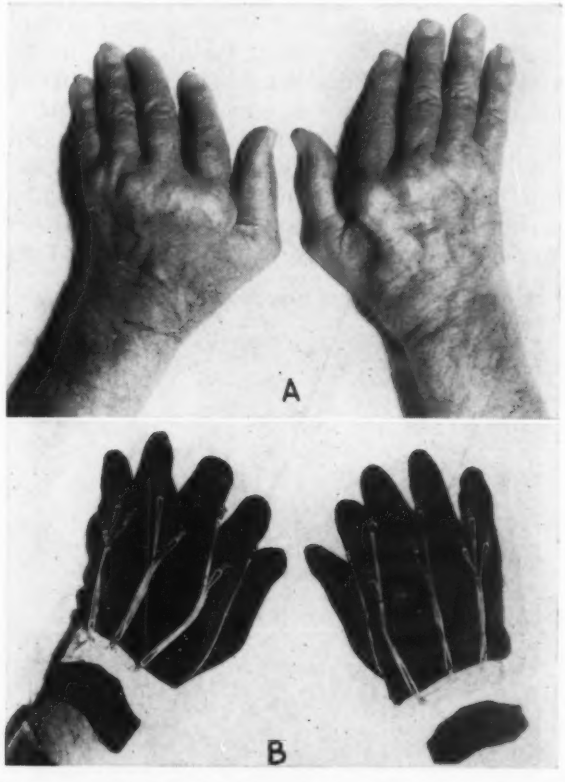


Fig. 5.—*A*, deformity of hands without assistive device. *B*, assistive device for correction of deformity of ulnar deviation. Note correction possible by proper application of tension.

2. *Orally Administered Procaine Solutions.*—The reported benefit following procaine infiltrations [9] or intravenously administered procaine solutions [10] inevitably led to attempted oral administration. Our own efforts to administer procaine hydrochloride orally in the solid form were abandoned when severe gastritis was frequently produced. However, when Roka and Lajtha [11]

7. Watkins, A. L.: Prescription of Physical Medicine in Office Practice, M. Clin. North America **31**: 1227 (Sept.) 1947.

8. Nesbitt, M. E.: Physical Therapy in the Treatment of Rheumatoid Arthritis, Phys. Therapy Rev. **29**: 345 (Aug.) 1949.

9. Travel, J.: Basis for the Multiple Uses of Local Block of Somatic Trigger Areas (Procaine Infiltration and Ethyl Chloride Spray), Mississippi Valley M. J. **71**:13 (Jan.) 1949. Cornell University Medical College: Treatment of Painful Disorders of Skeletal Muscle, New York State J. Med. **48**: 2050 (Sept. 15) 1948.

10. Graubard, D. J.; Kovacs, J., and Ritter, H. H.: The Management of Destructive Arthritis of the Hip by Means of Intravenous Procaine, Ann. Int. Med. **28**: 1106 (June) 1948.

11. Roka, G., and Lajtha, L. G.: Abolition of Pyloric Spasm by Orally Administered Procaine Solutions, Brit. M. J. **1**: 1174 (May 20) 1950.

demonstrated the feasibility of oral administration of solutions of procaine, this method was again tried. Current dosage schedules call for 5 cc. of a 5% aqueous solution of procaine hydrochloride three to four times daily. The bitter taste may be disguised more or less effectively by ginger ale or grape juice. We have not investigated the problem of vehicles with any degree of thoroughness, since the majority of patients prefer no vehicle, employing simply moderate dilution of the solution followed by additional water. This solution has been administered not only to this group but to more than 50 patients treated in the arthritis outpatient clinic of the University of Kansas Medical Center. In this group no gastritis has been noted. In fact, many patients have commented that salicylates no longer "upset their stomach" as much, appetite improves, and weight gain occurs.

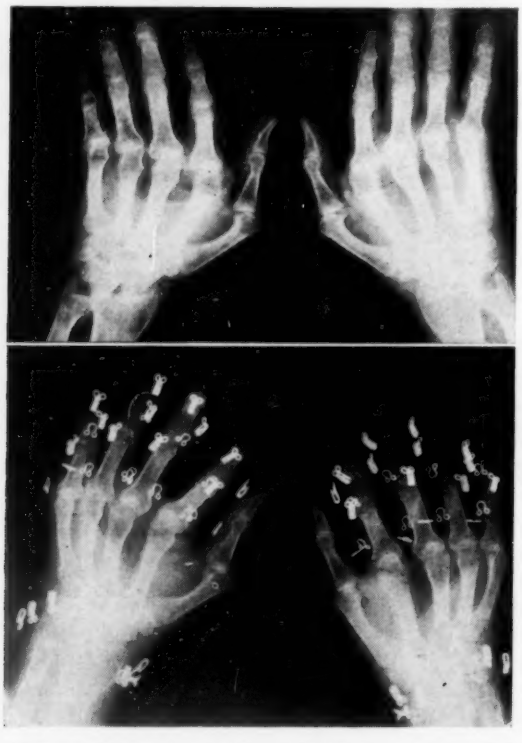


Fig. 6.—X-ray views, same positions as in figure 5. Note correction of ulnar deviation and decrease in subluxation at metacarpophalangeal joints.

Definite analgesia occurred in the majority of instances, beginning 15 to 20 minutes after ingestion and continuing for approximately four hours, and was of a quality described by some patients as comparable to heavy doses of salicylates without their side-reactions. The analgesic effect appeared to diminish with continued administration but persisted with intermittent dosage. For the latter reason, the solution is usually administered arbitrarily for three successive days, then omitted for the next four days; the drug is thus administered approximately half the time. The exact mode of action of the analgesic effect is unknown.[12] It is presumed to be hydrolyzed in the duodenum, at least as far as the paraaminobenzoic acid and diethylaminoethanol components are concerned, after oral ingestion. Neither of these latter two compounds have any striking analgesic action.[13] Preliminary studies on those patients receiving procaine solution orally indicate an absence of effect on the hemogram, including the quantitative eosinophil count and the erythrocyte sedimentation rate, and urinalysis demonstrates no alterations in the routinely examined components.

3. *Increased Attention to Nutritional Requirements.*—Obviously, no procedure will supply the deficiency in muscle bulk noted in figures 4 and 5 in the absence of an adequate nutritional balance. Although no one seriously believes that dietary regulation alone alters the course of rheumatoid arthritis, there is general agreement as to the necessity for what is vaguely termed an adequate and nutritious food intake. It has been shown [14] that severe injury or disease involving the musculoskeletal system incurs a negative nitrogen balance, which cannot be wholly corrected by dietary means or parenterally administered protein hydrolysates. Recent evidence [15] seems to indicate that testosterone propionate therapy may succeed in this respect in those patients with acute spinal cord injury. As Hellebrandt [16] has pointed out, full rehabilitation would seem impossible so long as tissue lost in the catabolic phase of invalidism was inadequately replaced by synthesis of new protein acquired through dietary channels. For this reason, we attempt to insure an adequate protein intake by insisting that each of our patients ingest a minimum of 3 oz. (85 gm.) of protein supplement in addition to his regular diet. This is prescribed in the form of one of the commercially available protein concentrates or as dehydrated skim milk reconstituted in skim milk or its equivalent, which is just as effective and less expensive. Supplementation of the diet by this means clinically appears to diminish fatigue and thus adds to the amount of work that the part can accomplish.

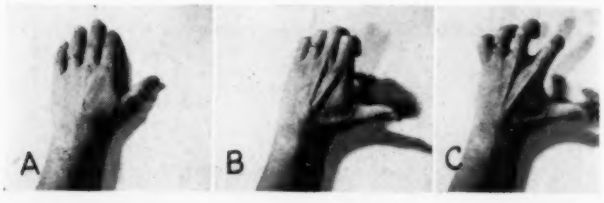


Fig. 7.—Routine exercises for the hand and fingers. *A*, resting position. *B*, finger flexion and adduction, hyperextension at metacarpophalangeal joints, radial deviation of hand and thumb. *C*, same as *B*, except for widespread abduction of flexed fingers.

COMMENT

A word should be spoken with respect to the terms rest and activity. Rest has been placed at the head of treatment procedures so often that the adjectives "absolute" and "inviolate" are being thought of in this con-

12. Isenberger, R. M.: The Pharmacologic Actions of Intravenous Procaine as an Analgesic Agent, Anesthesiology **10**: 343 (May) 1949.

13. Graubard, D. J., and Peterson, M. C.: Intravenous Use of Procaine in the Management of Arthritis, J. A. M. A. **141**: 756 (Nov. 12) 1949. Papper, E. M.; Brodie, B. B.; Lief, P. A., and Rovenstine, E. A.: Studies on the Pharmacologic Properties of Procaine and Di-Ethyl-Amino-Ethanol, New York State J. Med. **48**: 1711 (Aug. 1) 1948.

14. Peters, J. P.: Problems of Nitrogen Metabolism, Federation Proc. **3**: 197 (Sept.) 1944. Armstrong, W. D.: Bone Metabolism, ibid. **3**: 201 (Sept.) 1944. Howard, J. E., and others: Studies on Convalescence; Nitrogen and Mineral Balances During Starvation and Graduated Feeding in Healthy Young Males at Bed Rest, Bull. Johns Hopkins Hosp. **78**: 282 (May) 1946.

15. Cooper, I. S.; Rynearson, E. H.; MacCarty, C. S., and Power, M. H.: Testosterone Propionate as a Nitrogen-Sparing Agent After Spinal Cord Injury, J. A. M. A. **145**: 549 (Feb. 24) 1951.

16. Hellebrandt, F. A.: Physical Medicine and Rehabilitation in the Prevention of Chronic Invalidism, J. A. M. A. **142**: 967 (April 1) 1950.

nection. There is nothing therapeutic about rest except insofar as it may avoid flare-ups of the disease process. A certain measure of activity is not only possible within these same limits but must be insisted on. From the timidity with which activity is permitted at times and the repeated caution to avoid pain or soreness in exercising the part, the process of rehabilitation might be a long one indeed. Not only is it important that there be an exercise-rest balance,[17] but as Hill and Holbrook [28a] have noted the term tolerance must be carefully explained to the patient so that he will not stop the exercises with the onset of pain. So far as the hand program is concerned, our patients expect pain and discomfort during the exercise or use period. If the pain does not persist for several hours or increased discomfort does not result from performing the same activities the succeeding day, the patient is urged to disregard such symptoms. By insistence on increased pain tolerance, transient flare-ups of increased activity have been weathered without halting the rehabilitation efforts.

The use of the assistive device described, even in its present crude form, has met with immediate acceptance by the patient. Several patients have made their own devices, which attests not only to the simplicity of construction but also to the factor of motivation, which is so important in the treatment effort. The device may be laundered with ease, the only precaution being to insure against wrinkling in drying. Two patients have objected to wearing the cotton glove in public but have been quite satisfied to utilize soft suede gloves under these circumstances. Initially the tension may be tolerated for 30 to 60 minute periods only. No effort is made to exceed these limits at any one time, but it is soon established by the patient that he is able to tolerate the tension for increasingly long periods before becoming aware of the joint soreness. All patients thus far have increased the range of tolerance for hand activities within one month after incorporating the assistive device principle into the treatment program.

SUMMARY

1. A hypothesis is advanced to explain the mechanical basis for the hand deformity of ulnar deviation of the fingers at the metacarpophalangeal joints, flexion at the metacarpophalangeal joints, hyperextension of the proximal interphalangeal joints, and flexion of the distal interphalangeal joints seen late in rheumatoid arthritis.

2. Physical medicine rehabilitation measures are described that can readily be incorporated into the home activities of the average patient.

3. The use of a simple assistive device is described whereby hand function in a more nearly normal position is possible.

4. The concurrent employment of pain-relieving, reaction-blocking, and nutrition-supplementing mechanisms as necessary parts of the treatment program is emphasized.

17. Comroe, B. I.: Common Mistakes in Handling of Patients with Arthritis and Allied Conditions, J. A. M. A. **127**: 392 (Feb. 17) 1945. Piersol, G. M., and Hollander, J. L.: The Optimum Rest-Exercise Balance in the Treatment of Rheumatoid Arthritis, Arch. Phys. Med. **28**: 500 (Aug.) 1947.

CLINICAL NOTES

EARLY PERFORATION OF INTERVENTRICULAR SEPTUM AFTER MYOCARDIAL INFARCTION

REPORT OF A CASE WITH ANTEMORTEM DIAGNOSIS

George D. Geckeler, M.D.
Daniel J. Marino, M.D.
and
John E. Gregory, M.D., Philadelphia

Perforation of the interventricular septum following myocardial infarction is a rare complication. Since 1845, when Latham [1] first described this phenomenon, few reports have been recorded in the literature. In Sager's [2] study in 1934, there were 18 instances of septal perforation. Weber,[3] in a review of both domestic and foreign literature up to 1942, recorded 16 additional cases, including one of his own, making a total of 34 to that date. By 1948, Fowler and Failey,[4] in an excellent review, were able to add 24 cases, a total of 58. More recently reports have increased in frequency,[5] so that, with the present paper, 66 cases of acquired septal perforation are recorded in the literature.

More uncommon than the finding of an acquired septal perforation at autopsy is the recognition of this condition clinically. In Weber's [3] report of 34 cases, antemortem diagnosis was correct in only six. By 1948,[4] clinical acumen was more acute, so that, among the 24 cases added by Fowler and Failey,[4] the condition had been properly diagnosed during life in 11. In the eight cases reported since 1948, including the present one, there was clinical recognition of the presence of an interventricular rupture in six. These figures indicate that, while the incidence of acquired septal defects is very low, the recognition of this complication of myocardial infarction during life is becoming commoner.

As has been pointed out by various authors, the diagnosis of perforation of the infarcted myocardium is not difficult. If, in a given patient who has had a recent

From the Departments of Cardiology and Pathology, The Hahnemann Medical College and Hospital.

Clinical Professor of Medicine (Dr. Geckeler), Instructor in Medicine (Dr. Marino), and Professor of Pathology (Dr. Gregory).

1. Latham, P. M.: Lectures on Subjects Connected with Clinical Medicine Comprising Diseases of the Heart, London, Longmans, Brown, Green and Longmans, 1845, vol. 2, p. 168.

2. Sager, R. V.: Coronary Thrombosis: Perforation of Infarcted Interventricular Septum, Arch. Int. Med. **53**: 140 (Jan.) 1934.

3. Weber, M. L.: Perforation of Interventricular Septum Following Infarction: Intravitam Diagnosis: Report of Case and Survey of Literature, Ann. Int. Med. **19**: 973, 1943.

4. Fowler, N. O., Jr., and Failey, R. B., Jr.: Perforation of Infarcted Interventricular Septum: Report of Two Cases, One Diagnosed Antemortem, Am. J. M. Sc. **215**: 534, 1948.

5. Bickerman, L. J., and Irons, E. E.: Myocardial Infarction Resulting in Interventricular Septal Perforation: Report of Case Diagnosed During Life, Ann. Int. Med. **31**: 918, 1949. Ford, R. V.: Perforation of Infarcted Interventricular Septum, with Partial Heart Block, New England J. Med. **240**: 8, 1949. Fowler and Failey.[4] Nareff, M. J.; Sklar, L. J.; Kelly, F. T., and Reuling, J. R.: Rupture of Interventricular Septum, New England J. Med. **243**: 431, 1950. Greiland, R.: Perforation of Infarcted Interventricular Septum Diagnosed Ante Morem, Acta med. scandinav. **133**: 1, 1949.

myocardial infarction, there develops a loud, rough systolic murmur over the lower part of the sternum, which had not been present previously, and, in addition, if a thrill can be felt in this area, the diagnosis of acquired septal perforation should be carefully considered. It was these findings, except for the presence of the thrill, with the electrocardiographic evidence of acute anteroseptal infarction, that lead to the diagnosis in our case.

REPORT OF A CASE

A 55-year-old white man, an engineer, was admitted to the hospital, for the first time, complaining of severe retrosternal pain of two and a half hours' duration. On the night of admission, while teaching an evening class, he was suddenly seized with severe vise-like pain beneath the sternum, which radiated to the left shoulder and down the left arm. In spite of a hypodermic injection of morphine shortly after the onset of symptoms, the pain increased in severity. In addition, he stated he perspired profusely. For the past 10 days prior to the onset of his present complaint, the patient affirmed he was being treated by

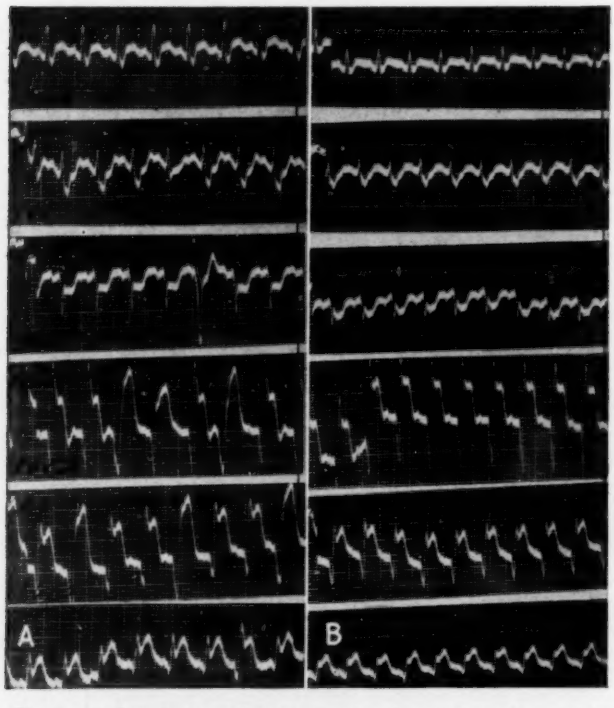


Fig. 1.—*A*, first electrocardiogram, showing sinus tachycardia and delay in intraventricular conduction time. *B*, second electrocardiogram. The RS-T segment in lead 1 is less elevated, the S waves are deeper in all the leads, and T waves are flat.

his private physician for an infection of the upper respiratory tract. There was no history of previous attacks of chest pain, either at rest or on effort, and there was no history of hypertension. The past history and family history were noncontributory.

Physical examination revealed that the patient was well-developed and well-nourished but in acute distress. He was extremely restless and in severe pain. His skin was cold and clammy, and there was slight cyanosis of the lips and the nail beds. There was no dyspnea or edema. His temperature was 98.3 F, pulse rate 92, and blood pressure 140/92.

The pertinent physical findings were related to the cardiovascular system. The left border of the heart was percussed just inside the midclavicular line in the fifth interspace. There was a normal sinus rhythm, at a rate of 92 per minute. There were no audible murmurs, no gallop rhythm, and no friction rub. The aortic second sound was present but not accentuated, and the pulmonic second sound was normal. There were a few moist crepitant rales present at the base of the right lung. There was no venous distention, and the liver was not palpable. Pulsations of the vessels of the extremities were normal. A diagnosis of myocardial infarction was made at this time.

On admission, the patient was given 15 mg. of morphine sulfate and 0.40 mg. of atropine sulfate by hypodermic injection. He was put at absolute bed rest and given oxygen by mask and 0.50 gm. of aminophylline in 200 cc. of 5% dextrose in water by intravenous infusion. Two hours after admission the patient continued to complain of retrosternal pain, but this was less severe than on admission. Examination at this time revealed sinus tachycardia, with a rate of 140, with frequent premature systoles, and with a definite gallop rhythm. No murmurs were heard, but moist crepitant rales were present at both lung bases. An electrocardiogram (fig. 1*A*) showed sinus tachycardia, with a rate of 135, and a delay in intraventricular conduction time. Frequent ventricular premature systoles were noted. The RS-T segment was elevated in lead 1 and all the precordial leads and sharply depressed in leads 2 and 3. There were deep Q waves in all the precordial leads. These findings indicated the acute stage of an anteroseptal myocardial infarction. Treatment at this time consisted of the administration of meperidine (demerol®) hydrochloride as required for pain, quinidine sulfate, and anticoagulants.

Nine hours after admission, one of us (G. D. G.) noted that the patient was slightly confused, restless, and cyanotic. His heart beat was regular (155 per minute) and there was a rough systolic murmur replacing the first sound, with maximal intensity along the lower left sternal border at about the fourth interspace; this murmur was also directly over the sternum at this level and to the right of the sternum. It ceased abruptly above the level of the fourth interspace on the right. This murmur had

Fig. 2.—Small stellate defect in the interventricular septum.

not been heard previously. It was felt that the murmur, along with the picture of collapse, represented a septal perforation, even though such a diagnosis was hazardous in view of the length of time after the onset of the coronary thrombosis.

A second electrocardiogram (fig. 1*B*) showed the RS-T segment in lead 1 to be less elevated, with deeper S waves in all of the limb leads and flat T waves; the premature ventricular systoles had disappeared, and there were no other significant changes.

Urinalysis showed a specific gravity of 1.018; the reaction was acid, and there was a distinct trace of protein. There was no sugar, and the result of the test for acetone was 1+. Microscopically, there were occasional coarse granular casts. The red blood cell count was 6,000,000, hemoglobin 16.6 gm., and white blood cell count 14,000, with 84% polymorphonuclear cells, 14% lymphocytes, and 2% monocytes. Blood urea nitrogen was 28 mg. per 100 cc. and blood sugar 65 mg. per 100 cc.

The patient's clinical condition showed progressive deterioration. He remained disoriented and in a state of collapse, with a fast heart rate and dropping blood pressure until, in the 16th hour after admission, no reading could be obtained. Pulmonary edema increased in degree, and two hours before death the patient coughed up blood-streaked sputum. He died 23 hours after admission, 25½ hours after the onset of symptoms.

At autopsy (Dr. F. E. Murphy) the heart was found to be moderately enlarged, owing to dilatation of both ventricles, most pronounced on the left. The heart weighed 340 gm. A massive infarct was seen over the anterior surface of the heart, which began 2 cm. from the coronary sulci and extended to the apex

of the left ventricle and involved almost the entire width of the septum. The most striking finding was a small stellate defect in the interventricular septum, 3 mm. in diameter (fig. 2), located in the anterior edge of the septum, 4 cm. above the apex of the right ventricle.

Examination of the coronary arteries revealed the anterior descending branch to be completely occluded at a point 0.5 cm. from its origin by a reddish yellow, slightly firm, adherent thrombus. This thrombus extended for approximately 1.5 cm. Beyond this point, the lumen was greatly reduced in size and lesser branches showed variable degree of narrowing but no complete occlusion. The circumflex branch showed moderate sclerosis and calcification, with narrowing of about 30% to 50%. The right coronary showed a slightly more marked degree of narrowing, but at no point was there complete occlusion. The heart valves were not remarkable. There was moderate to marked arteriosclerosis of the aorta. The lungs were heavy, weighing 890 and 580 gm., for the left and right, respectively, with marked congestion, edema, and bloody froth in the tracheobronchial tree. Microscopic examination of tissue near the septal perforation showed an unusually large number of polymorphonuclear leukocytes (fig. 3). The muscle fibers contained no recognizable nuclei and were fragmented, but they still showed definite cross striations. There was no evidence either grossly or microscopically of hemorrhage or scarring in the septum. Sections taken elsewhere in the myocardium showed no scarring, despite the considerable degree of coronary sclerosis.

COMMENT

The most interesting feature of this case, and the major reason for our reporting it, is the very early occurrence of the septal perforation. The diagnosis was made 12 hours after the onset of symptoms. In the series of 72 cases of myocardial perforation reported by Edmondson and Hoxie,[6] the average time of rupture was 7.4 days after onset, 78% occurring between the 3rd and 12th days. Six instances of rupture one day after infarction were noted by these authors. Mallory, White, and Salcedo-Salgar,[7] in their exhaustive study of the development and healing of myocardial infarcts, observed one case of rupture occurring one day after infarction and cited one similar case from Levine.[8] In their own series plus that of Levine,[8] a total of 17 cardiac ruptures, Mallory and co-workers [7] found that 65% occurred in the first week and 29% in the second week after infarction. The only recorded case of perforation that occurred earlier than the one herein reported was that of Hyman,[9] in which rupture of the interventricular septum was diagnosed four hours after the onset of symptoms of infarction. Death occurred in this patient 5½ hours after the first symptoms, 1½ hours after perforation was noted. In our own case, the patient died 25½ hours after the initial symptoms, or 13½ hours after diagnosis of perforation. These findings indicate the serious nature of rupture of the infarcted septum. In the literature cited above, the survival time in more than 50% is less than seven days. It must be noted, however, that Wood and Livezey [10] have reported an unusual case, with survival for almost five years.

Since rupture of infarcted myocardium is a relatively uncommon complication, the reasons for its occurrence have stimulated considerable thought. Edmondson and Hoxie [6] and Mallory, White, and Salcedo-Salgar [7] comment at some length on the factors thought to play important roles. These factors consist of hypertension, which increases the stress on the weakened wall; well-developed necrosis, the soft necrotic tissue being markedly less resistant; polymorphonuclear infiltration, since dying and dead leukocytes release proteolytic enzymes, leading to softening of the tissue; hemorrhage into the dying or dead myocardium; and, finally, the absence of scar tissue, which would obviously be more resistant. In Hyman's report,[9] there is no mention of blood pressure and no microscopic description is included, but the gross examination revealed that the perforation occurred in an area of myocardial hemorrhage.

In our own case there is, as has been mentioned, an excess number of polymorphonuclear leukocytes and an absence of scarring. Furthermore, the blood pressure was 140/92 after infarction and before perforation occurred. While there was no knowledge of previous hypertension, it may be safely said that, in a patient with as massive an infarct as was present in our patient, these

Fig. 3.—Photomicrograph of tissue near the septal perforation, showing polymorphonuclear leukocytes.

levels are higher than are usually encountered. The postmortem findings, which revealed the weight of the heart to be only 340 gm., with absence of left ventricular hypertrophy, was in keeping with the history of normotension. Thus, in this case there was what might be called "relative" hypertension, associated with the excess leuko-

6. Edmondson, H. A., and Hoxie, H. J.: Hypertension and Cardiac Rupture; Clinical and Pathologic Study of 72 Cases, in 13 of Which Rupture of Interventricular Septum Occurred, Am. Heart J. **24**: 719, 1942.

7. Mallory, G. K.; White, P. D., and Salcedo-Salgar, J.: The Speed of Healing Myocardial Infarction: A Study of the Pathologic Anatomy in Seventy-Two Cases, Am. Heart J. **18**: 647, 1939.

8. Levine, S. A.: Coronary Thrombosis: Its Various Clinical Features, Medicine **8**: 245, 1929.

9. Hyman, A. S.: Spontaneous Rupture of Heart: Perforation of Interventricular Septum, Ann. Int. Med. **3**: 800, 1930.

10. Wood, F. C., and Livezey, M. M.: Five-Year Survival After Perforation of Interventricular Septum Caused by Coronary Occlusion: Histologic Study of Kidneys After 350 Injections of Mercurial Diuretics, Am. Heart J. **24**: 807, 1942.

cytes and an absence of scarring, three of the five factors mentioned above. An additional factor worth consideration in the production of rupture in this case was the early onset of a severe degree of cardiac failure. This complication of acute left ventricular failure occurred shortly after the patient's admission to the hospital, approximately seven hours prior to the clinical suspicion of interventricular perforation. During this period, the blood pressure at the level described above was maintained. The compensatory mechanisms that result in an increase in the systolic and diastolic volume of blood in the left ventricle, plus the increase in intraventricular pressure, added to the previously mentioned factors, may well have been the most important factors in the development of interventricular rupture. It should be pointed out that, if the factors tending to produce rupture are present and if the patient is seen after the first 12 hours, clinical evidence of septal rupture may be encountered on first examination. Obviously, early rupture represents the early development of factors leading to disintegration of the wall, or the concomitant presence of several of them, or the activity of one or more of them to an unusually marked degree.

SUMMARY

A case of perforation of the interventricular septum 12 hours after onset of symptoms of myocardial infarction is reported. It is pointed out that clinical recognition of this complication during life is not difficult and is being accomplished with increasing frequency. Possible factors in early perforation are discussed.

230 N. Broad St. (Dr. Geckeler).

PNEUMATOSIS OF THE COLON

Richard H. Marshak, M.D.
Joan J. Lipsay, M.D., New York
and
A. I. Friedman, M.D., Hackensack, N. J.

Pneumatosis intestinalis may be defined as the intramural presence of gas within the intestinal tract. It has previously been recorded under various descriptive titles: "intestinal emphysema," "emphysema of the cecum," "gas cysts of the intestine" (pneumatosis cystoides intestini), and "pseudocysts." The case to be presented is unusual in that it involved the left side of the colon and the symptoms developed with dramatic suddenness after sigmoidoscopy. In view of the rectal pathological condition seen by the sigmoidoscopist, the possibility that the pneumatosis of the colon predated his examination, though unlikely, cannot be excluded.

From the Radiologic and Gastroenterologic Departments of Mount Sinai Hospital, New York.

1. Bang, B. L. F.: Luftholdige kyster i väggen af ileum og i nydannet bindeväv pä sammes serosa, Nord. med. Ark. **8**: 1, 1876; cited by Jackson, J. A.: Gas Cysts of the Intestine, Surg., Gynec. & Obst. **71**: 675-678, 1940. Feldman, M.: Clinical Roentgenology of the Digestive Tract, ed. 3, Baltimore, The Williams & Wilkins Company, 1948, p. 418.
2. Moore, R. A.: Intestinal Pneumatosis, Am. J. Dis. Child. **38**: 818, 1929.
3. Nitch, C. A. R., and Shattock, S. G.: Diffuse Emphysema of Intestinal Wall (Two Cases) with Remarks upon Pneumatosis, Proc. Roy. Soc. Med. (Section of Pathology) **12**: 46, 1919.
4. Lerner, H. H., and Gazin, A. I.: Pneumatosis Intestinalis: Its Roentgenologic Diagnosis, Am. J. Roentgenol. **56**: 464, 1946.

REPORT OF A CASE

Mrs. E. A., 55, was seen by her physician because of rectal bleeding of three weeks' duration. Her past history was noncontributory for tuberculosis, diabetes, and intestinal disease. An appendectomy had been performed 25 years previously. Rectal examination revealed internal hemorrhoids, but sigmoidoscopy was performed to eliminate other possible causes of rectal bleeding. The rectosigmoid was covered by a thin, gray exudate, overlying a reddish, slightly hypertrophic mucosa that resembled granulation tissue. The operator was impressed by the unusual granular mucosa in this region. Immediately after the removal of the instrument, the patient complained of severe, diffuse, abdominal pain. The pain was of such intensity that she was admitted to the hospital. The abdomen was moderately tense, with tenderness over the descending colon. The sigmoid could be palpated as a doughy mass. Laboratory examination disclosed no abnormalities. A barium enema was performed. The first was unsuccessful; the second one revealed the typical picture of pneumatosis of the colon. The lumen of the colon was narrowed from the sigmoid to the distal transverse colon and compressed by huge intramural air pockets, leaving only a thin streak of barium. There was no other abnormality throughout the entire large bowel. There was no evidence of any free air under the diaphragm (fig 1). The symptoms of abdominal pain and constipation remained intense for a period of one week and then gradually decreased. She was discharged from the hospital and seen intermittently thereafter. As late as six months after the original trauma, symptoms of abdominal distress persisted, with intermittent constipation and a sense of fulness following evacuation. X-rays taken at this time continued to display the intramural, hugh, round air sacs compressing the lumen of the descending colon (fig. 2).

COMMENT

The cause of pneumatosis intestinalis has been variously identified. Some have expressed the belief that it is due to gas production by bacteria,[1] but organisms have rarely been identified on culture. Moore [2] has advanced the theory that it is the result of mechanical phenomena due to a break in the continuity of the mucosa in association with increased intraluminal pressure. The condition may follow ulceration of the mucosal wall. The condition in more than 55% of the 85 cases of pneumatosis reported by the British investigators, Nitch and Shattock,[3] was associated with duodenal or gastric ulcer. Most instances of pneumatosis of the large bowel have been reported to occur in the cecum, in association with ulcers at the base of the appendix.[4]

The gross pathological condition is generally dependent on the location and the degree of air infiltration. These air pockets take the form of discrete, localized or diffuse vesicles along the course of the bowel. They usually appear sessile but may be pedunculated, sausage-shaped, or spherical. There is no characteristic histopathological picture, though some authors have described giant cell formation, presumably the result of a chronic, granulomatous, inflammatory process. The gas appears to be forced into the wall of the bowel, either submucosal or subserosal, and dissects its way beneath the serosal layer, limited only by the peritoneal attachments. Occasionally this dissection takes place beneath the layers of peritoneum, to lodge subdiaphragmatically. It may be important to consider this condition in the etiology of spontaneous pneumoperitoneum when the cause is not apparent. Finally, as the gas accumulates in the wall of the bowel (in children the gas pockets are more commonly found in the submucosa and in adults in the subserosa),[2] it produces an invagination of the mucosal or muscular coats with a reduction in the diameter of the

lumen, resulting in partial or even complete obstruction. Jackson [5] has described a case in which volvulus developed as a complication of pneumatosis of the small bowel.

The symptoms usually have been those of incomplete intestinal obstruction, namely, colicky pain and constipation; more frequently than not, the symptoms of the primary condition overshadow those of the pneumatosis, so that the latter may be unrecognized. On physical examination, one may palpate a boggy tumor in either lower abdominal quadrant. The bowel is tender and may feel "spongy." Some [6] have described a characteristic change in position of the tumor from day to day. The diagnosis has ranged from appendicitis and appendiceal abscess to carcinoma. In many cases surgical exploration was done because the symptoms and signs were pathognomic of peritoneal irritation. At operation the intestine exhibits grayish, rounded, crepitant, yielding masses where the gas cysts "balloon out" the surface of the intestinal wall. Burt [7] reported a case of pneumatosis of the left half of the colon, associated with subcutaneous emphysema, following excision of an anorectal fistula. Postoperatively his patient showed signs of peritoneal irritation and emphysema extending over the entire left portion of the abdomen and the lower part of the chest. A laparotomy was performed, and free air was encountered in the subcutaneous tissue, subperitoneal tissue, and mesentery of the entire left side of the colon.

The x-ray picture is typical. Areas of increased translucency (filling defects) lie between the lumen (outlined by the contrast medium) and the outer margin of the intestinal canal and may extend for variable distances along the bowel. It is impossible to fill out completely the lumen of the bowel, because of the pressure exerted by the tense

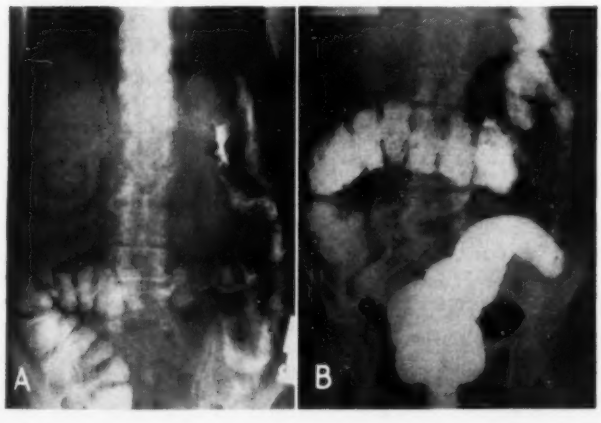


Fig. 1.—*A*, barium enema examination reveals a narrowed eccentric lumen compressed by huge air pockets extending from the sigmoid to the distal transverse colon. *B*, the lumen of the descending colon is more distinctly narrowed, but barium passes readily into the remainder of the large bowel.

gas cysts. These filling defects are inconstant and variable in size and often shift their position daily. Frequently, the lumen is compressed concentrically and the contrast medium appears as a thin, irregular stream in the midline of the colon, with air shadows extending on either side of the bowel silhouette.

The treatment is conservative unless complete obstruction supervenes. As a rule the gas is absorbed after a variable period of time, with disappearance of all symptoms. There are available several reports of patients with pneumatosis intestinalis diagnosed by x-ray examination in whom evidence of the earlier condition was not visualized in subsequent surgery for other causes.

Fig. 2.—Reexamination six months later still reveals the large air pockets involving the left colon. The lumen is narrowed, and the air cysts create a scalloped effect on the barium column. There is little change in the appearance of the pneumatosis as compared with that in the previous examination.

SUMMARY

A case of pneumatosis of the colon with the characteristic symptoms and roentgen picture, involving the left side of the colon, is reported. In view of the possibility that the onset of the pneumatosis in this case was induced by sigmoidoscopy, we wish to suggest trauma as an etiological agent in the production of this syndrome.

1075 Park Ave. (Dr. Marshak).

5. Jackson, J. A.: Gas Cysts of the Intestine, Surg., Gynec. & Obst. **71**: 675-678, 1940.
6. Nitch and Shattock,[3] Jackson.[5]
7. Burt, C. A. V.: Emphysematous Acute Abdomen and Subcutaneous Emphysema from Intrarectal Operation, Ann. Surg. **130**: 277, 1949.

Megaloblastic Anemia in Infancy.—Megaloblastic anemia in infancy is a nutritional anemia which responds specifically to folic acid. It is one of the last entities to be separated from the ill defined syndrome of von Jaksch's anemia. It occurs between the fifth and eleventh month, as a rule, and is due either to a deficiency of folic acid in the diet or to an inability of the infant to absorb and utilize this vitamin. Human milk and goat's milk contain less folic acid than cow's milk and infants fed exclusively on human or goat's milk are more apt to develop this syndrome, particularly if there is also a deficiency of ascorbic acid in the diet. The response to oral administration of folic acid is prompt. Ascorbic acid may also be required if a deficiency exists. Vitamin B_{12} is less effective than folic acid. The best regimen for control of this anemia is obtained by the oral administration of folic acid 15 mg. daily and ascorbic acid 100 mg. daily. In severely ill babies, liver extract—2 cc.—may be used intramuscularly as well.—Mila I. Pierce, Management of Anemia in Childhood, *Postgraduate Medicine*, January, 1952.

TALC PNEUMOCONIOSIS

Paul S. Friedman, M.D.
Milton A. Bell, M.D.
and
Leon Solis-Cohen, M.D., Philadelphia

There is increasing evidence that occupational inhalation of pulmonary irritants is one of the major causes of pulmonary fibrosis. Free silica is a common etiologic agent. Talc, a hydrous magnesium silicate, is an unusual cause for such pneumoconiosis. The following case demonstrates the roentgen and clinical characteristics of talc pneumoconiosis.

REPORT OF A CASE

The patient, a 65-year-old white man, was referred for roentgen study of the chest on June 19, 1950, because of a chronic cough which was intensified during the preceding week. He had a cough of many years' duration accompanied by production of frothy sputum. On June 9, 1950, sneezing, coryza, intensified cough, and purulent postnasal discharge and expectoration developed. There was accompanying onset of exertional dyspnea. Walking three blocks caused dyspnea.

For a period of 24 years since 1926, he had been employed in the handling and packaging of elastic cord (rubber thread) coated with talc powder. In the handling of this rubber, he removed rubber ribbons, heavily coated with talc, from their containing box. He fed one end of the rubber into a reed that separated the strands; the rubber strands then passed onto a spindle which made skeins or cones of rubber thread. From the moment the containing box was opened, the atmosphere of the working

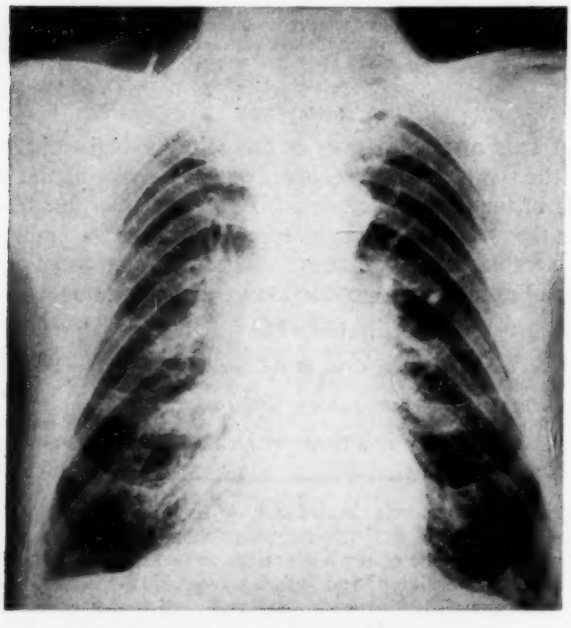


Fig. 1.—Chest, posteroanterior projection. This demonstrates diffuse pulmonary fibrosis, pulmonary emphysema, pleural thickening, and talc plaque in right upper hemithorax.

area was cloudy with talc powder. No exhaust or ventilating fans were present. In 1914 a right thoracotomy for empyema was performed.

Physical examination on June 9 revealed a thin, white man with a temperature of 99.4 F, a pulse rate of 112, and blood pressure of 110/74. There was mucopurulent nasal secretion. There was a thoracotomy scar over the right lower posterior hemithorax. Chest expansion was restricted. Coarse moist rales as well as areas of bronchial breathing were present throughout both lungs. No clubbing of the fingers was present. The patient was treated with bed rest. He received penicillin orally, intramuscularly, and by nose drops.

On June 13, he was clinically improved and returned to work. Temperature and pulse rate were normal. Cough was less prominent, but dyspnea was still marked on walking four blocks or uphill. There was minimal night cough when he was recumbent.

Fig. 2.—Chest, lateral projection. Note increased anteroposterior diameter accompanying the pulmonary emphysema, foci of pulmonary calcification.

There were momentary episodes of respiratory obstruction. Coarse moist rales were still heard over the lower lobe of the right lung. Fluoroscopic and radiographic study of the chest on June 19 revealed marked reduction in thoracic respiratory mobility. There was reduced diaphragmatic excursion during respiration.

Lungs.—There was evidence of diffuse pulmonary emphysema, characterized by low position of the diaphragm and increased anteroposterior thoracic diameter. There was diffuse linear intensification of the pulmonic markings owing to pulmonary fibrosis. This was most prominent in the right lung. Stable foci of calcification due to previous tuberculous infection were visualized in the upper lobe and hilar region of the left lung (fig. 1 and 2).

Pleura.—There was thickened pleura present, most prominent on the right, where it produced stragihtening of the right hemidiaphragm. A linear homogeneous opacity, 9 cm. in length and 1 cm. in width, was demonstrated in the upper right hemithorax. Its axis was obliquely vertical, and it apparently represented a talc pleural plaque.

Heart and Mediastinal Structures.—There was no abnormal mobility during respiration. The heart was not measurably enlarged. There was increased convexity of the left cardiac border owing to prominence of the right ventricle. Elongation of the aorta due to arteriosclerosis was present. The sputum was negative for tubercle bacilli.

A mixed catarrhal vaccine series was administered in an attempt to improve immunity to respiratory pathogens. Penicillin has been used periodically for acute episodes of tracheobronchitis with purulent secretions. The patient has been relatively comfortable though not entirely free of cough.

An electrocardiogram on Feb. 13, 1951, revealed low amplitude of the QRS component in the limb leads. It was otherwise natural.

COMMENT

Talc pneumoconiosis has been described in talc miners and millers. Since talc is used industrially as a filler, as a dusting powder, and as an absorbent, the possibility of excessive exposure is not a limited one. Bethene, in 1935, experimentally demonstrated its irritant characteristics when pleural adhesions were caused in cats and dogs by the intrapleural insufflation of talc powder. Since the free silica content of talc is 0.5% or less, its irritant characteristics are apparently not a result of its free silica content.

Clinically, talc pneumoconiosis usually caused dyspnea, chronic cough, chest pain, and fatigue. The dyspnea is usually more intense than the roentgen manifestations of fibrosis would suggest. Limited chest expansion is invariably present. The patients are usually thin, minimally cachectic in appearance, and in 70% of cases exhibit curving nails or clubbed fingers. The patient described herein exhibited all of these changes except clubbing of the fingers.

The roentgen changes resulting from talc pneumoconiosis are characterized by fibrosis, emphysema, pleural thickening, pleural and subpleural plaques ("talc plaques"), and a minimally increased incidence of tuberculous infection. The fibrosis is of a fine linear and granular character, similar in type to that visualized in asbestosis. It tends to be most intense in the midportions of the lungs, extending into the basilar segments of the lower lobes. A moderate degree of diffuse pulmonary emphysema is present. Partially owing to the emphysema and partially owing to the pulmonary fibrosis, there is a prominence of hilar and intrapulmonic segments of the pulmonary arteries.

The pleura exhibits diffuse thickening and fibrosis. In addition, linear areas of pleural density, so-called talc plaques, occur. These were seen in 6.3% of all talc miners.[1] They occur in the peripheral and subpleural aspects of the visceral pleura. These may vary from single, linear, homogeneous, sharply marginated opacities, several centimeters in length, to irregularly shaped, extensive opacities of varying density. It is felt these occur secondary to accumulation of talc in peripheral lymphatics with lymphatic dilatation and stasis, associated with calcium localization.

There has been suggestive statistical evidence of increased susceptibility to significant tuberculous infection.

SUMMARY

A case of talc pneumoconiosis is reported with typical clinical and roentgen characteristics. This report seemed indicated in order to direct attention to talc as an occupational hazard and to the probable significance of other inhaled pulmonary irritants as causative agents in unexplained pulmonary fibrosis.

1422 Chestnut St. (Dr. Friedman).

1. Greenburg, L.: The Dust Hazard in Tremolite Talc Mining, Yale J. Biol. & Med. **19**: 481-501 (March) 1947.

BRONCHOGRAPHY IN IODINE SENSITIVITY

Peter A. Theodos, M.D., Philadelphia

The instillation of iodized oil into the bronchial tree is widely used in the diagnosis of bronchiectasis, the delineation of distorted or obstructed bronchi, and the determination of communications between bronchi and the pleura, skin, or air cysts. All the radiopaque preparations commonly used in bronchography today contain iodine. Those most frequently used are lipiodol,® which contains 40% iodine in poppyseed oil, and iodochloral,® which contains 27% iodine and 7.5% chlorine in organic combination with highly refined peanut oil. Bronchography with these agents is usually innocuous, but untoward reactions, such as iododerma and asthma-like reactions with fatal outcome in two cases, have been reported.[1] It is imperative, therefore, that proper inquiry be made as to sensitivity to iodine prior to the use of any iodine-containing contrast medium.

A patient with suspected bronchiectasis was recently admitted to the hospital for study. The presence of pronounced iodine sensitivity made the use of iodized oil as a contrast medium for bronchography inadvisable and even hazardous. However, since the use of corticotropin (ACTH) had been reported in the treatment of sensitivity to iodine,[2] it was decided to carry out the iodized oil study after preliminary desensitization with corticotropin. A satisfactory bronchogram with minimal reactions was obtained. Because of this favorable outcome, the following report is considered of interest:

REPORT OF CASE

I. O., a 46-year-old white woman, was first seen in October, 1950, because of severe paroxysmal cough, copious foul expectoration, and pain in the left lower part of the chest posteriorly. A diagnosis of suspected bronchiectasis had previously been made, in 1944, after an iodized oil study. At that time severe swelling of the submaxillary glands developed, along with "blotches all over the body resembling strawberry hives," which lasted 10 days after the bronchogram. Subsequent bronchograms, in 1945 and 1947, again produced reactions manifested by swelling of the submaxillary glands, fever, and hives. Although these studies revealed equivocal evidence of bronchiectasis in the lower lobe of the left lung, a segmented resection was done in 1947. Symptoms were then few, until November, 1948, when cough recurred. This had become progressively severer and was productive of several cups of foul sputum daily at the time of the patient's admission to the hospital.

Physical examination disclosed nothing essentially abnormal except in the lungs, where there was impairment of percussion and distant breath sounds over the lower lobe of the left lung. No rales were heard.

Fluoroscopic and roentgenographic examinations revealed portions of the posterior segments of the 9th and 10th ribs to be missing. The left side of the diaphragm was slightly elevated

From the Barton Memorial Division and the Department of Medicine, Jefferson Medical College Hospital.

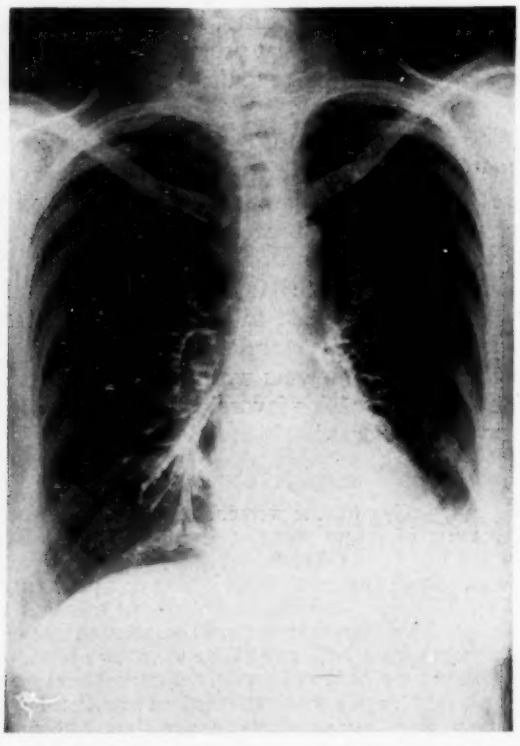
1. Belote, G. H.: Iododerma from Iodized Oil, J. A. M. A. **89**: 882, 1927. O'Donovon, W. J.: Intratracheal Injection of Lipiodol: Generalized Iodide Eruption: Death, Brit. M. J. **2**: 935, 1927. Carmichael, D. A.: Iodine Poisoning and Iodism from Lipiodol, Canad. M. A. J. **26**: 319, 1932. Scadding, J. G.: Acute Iodism Following Lipiodol Bronchography, Brit. M. J. **2**: 1147, 1934. Goldstein, D. W.: Fatal Iododerma Following Injection of Iodized Oil for Pulmonary Diagnosis, J. A. M. A. **106**: 1659, 1936.

2. Ruh, A. R.: Hypersensitivity in Disease, with Special Reference to Periarteritis Nodosa, Rheumatic Fever, Disseminated Lupus Erythematosus and Rheumatoid Arthritis, Harvey Lect. **42**: 106, 1946-1947.

and moved sluggishly. The heart was slightly deviated to the left. A minimal degree of scattered fibrosis was present in both lungs.

Bronchoscopic examination revealed the mucosa to be moderately inflamed throughout. The bronchial orifices were all easily visualized and were all normal in contour. It was not possible to demonstrate the bronchial stumps or any evidence of a bronchopleural fistula. A small amount of greyish secretion was aspirated from both bronchial tubes, which revealed no acid-fast bacilli on smear and culture.

A blood cell count revealed hemoglobin 12.5 gm., red blood cells 3,950,000 per cubic millimeter, white blood cells 10,000 per cubic millimeter, segmented neutrophils 50%, lymphocytes 40%, monocytes 6%, and eosinophils 4%. The urine was normal except for a specific gravity of 1.010. Four plain smears of the sputum, which measured 30 to 127 gm. a day, revealed no acid-fast bacilli. Culture of the bronchial secretions revealed Streptococcus viridans, Streptococcus nonhemolyticus, Staphylococcus albus, and nonpathogenic Neisseria.



Bronchogram obtained after desensitization with corticotropin (ACTH). There were no abnormalities except for minimal cylindrical bronchiectasis of the posterior segment of the lower lobe of the left lung.

The presence of a bronchopleural fistula or residual bronchiectasis was suspected, and an iodized oil study was advised. The patient, however, refused to have this done, because of her previous reactions and because she had been warned by several physicians that she was sensitive to iodine and might die from the reaction. Tests for iodine sensitivity were performed. A scratch test was made, with the use of one drop of chloriodized oil (iodochloral®) on the flexor surface of the left arm. No reaction was noted within 48 hours. The test was repeated, with 0.1 cc. given subcutaneously. Slight elevation and reddening of the injected area were noted in 48 hours. Several days later the patient was given 5 minims (0.30 cc.) of a saturated solution of potassium iodide orally. Marked swelling of the submaxillary glands developed in seven hours and lasted four days. At the same time she complained of malaise and manifested a few red blotches on her arms, which persisted for 48 hours.

Desensitization with corticotropin was then carried out. The patient was given 20 mg. twice a day for four days and then 10 mg. daily for five days. On the third day of therapy a bronchogram, with chloriodized oil, was attempted by the gravity method. No oil was gotten into the bronchi, owing to an irritable throat and laryngeal spasm. However, about 10 cc. of the oil was swallowed. Very slight swelling of the submaxillary glands developed in five hours, which lasted several hours. The next day a catheter was inserted through a laryngoscope and 15 cc. of chloriodized oil injected into both lungs under fluoroscopic guidance. Excellent visualization of the entire bronchial tree was obtained, with normal findings except for minimal cylindrical bronchiectasis of the posterior segment of the lower lobe of the left lung (see accompanying figure). Only a slight reaction was experienced by the patient, which lasted several hours and which in no way compared with those she had previously experienced. Repair of the operative scar and excision of the lower lobe of the left lung was subsequently carried out successfully, with considerable symptomatic relief and alleviation of the pain.

COMMENT

The infrequency of severe reactions following iodized oil studies is probably due to the stable chemical combination between the iodine and the unsaturated fats in the oil. Elimination of the iodine from the body is effected mechanically through the cough reflex and by absorption through the usual body channels. Most of the oil is generally expectorated within three hours. Little decomposition of the oil occurs in the lung, but rapid breakdown and absorption of the liberated iodine occurs in the gastrointestinal tract. Various authors have reported that the toxic and allergic phenomena occur only when the oil reaches the gastrointestinal tract. It seems likely, therefore, that severe reactions in patients allergic to iodine may occur if the iodized oil is swallowed during or shortly after bronchography.

The effectiveness of corticotropin therapy in enabling a sensitive patient to tolerate the use of iodized oil is well exemplified in this case. The exact mechanism of action has not been established but is probably related to the corticotropin stimulation of the adrenals. This allows the cells of the body to function in a relatively normal physiological manner, despite the hypersensitivity that is inhibited or reversed. With the small doses necessary to effect desensitization, the commonly reported side-effects and complications to such hormonal therapy need not be feared. It will be possible, therefore, to carry out bronchographic study successfully and safely in the face of what previously has been considered a distinct contraindication.

SUMMARY

A case of iodine sensitivity is reported in which bronchographic study with an iodine-containing contrast medium was successfully carried out after desensitization with corticotropin (ACTH).

1930 Chestnut St.

Treatment of Breast Abscess.—The best—easily the best, and perhaps the only—way to treat a breast abscess is to make a radial incision as soon as the overlying skin becomes oedematous. The incision need be no longer than is required to insert the index-finger, which then breaks down the adhesion; and a small soft drain is inserted into what should at this stage be a relatively small and simple cavity. Parenteral penicillin is prescribed to exorcise the surrounding cellulitis, and in nineteen cases out of twenty the abscess heals without more suffering to the patient than is occasioned by introducing thiopentone into a vein. In the treatment of diseases of the breast the hollow needle, whether to aspirate abscesses, to puncture cysts, or in cases of suspected carcinoma to perform what is termed a "drill biopsy," had better be left to those who have a particular skill, a peculiar disposition, or a poverty-stricken comprehension.—H. J. B. Atkins, Pain in the Breast, *Lancet*, Feb. 9, 1952.

COUNCIL ON FOODS AND NUTRITION

The following products have been accepted as conforming to the rules of the Council on Foods and Nutrition.

JAMES R. WILSON, M.D., *Secretary.*

Armour and Company, Chicago, Packer; Gerber Products Company, Fremont, Michigan, Distributor.

GERBER-ARMOUR CHOPPED PORK is made from meats (trimmed of fat, sinews and connective sheaths) with added salt, prepared under inspection of the Meat Inspection Division of the United States Department of Agriculture.

Analysis (submitted by manufacturer).—Total solids 27.9%, moisture 72.1%, ash 1.5%, fat 6.5%, protein (N × 6.25) 19.7%, carbohydrates (none), crude fiber (none), and salt 0.5%.

Vitamins and Minerals	Mg. per 100 Gm.
Thiamine	0.158
Riboflavin	0.246
Nicotinic acid	2.45
Pyridoxine	0.28
Calcium	10
Phosphorus	133
Iron	0.9

Calories.—1.38 per gram; 39.1 per ounce.

Use.—For use in the feeding of older infants and young children.

Beech-Nut Packing Company, Canajoharie, New York.

BEECH-NUT COOKED CORN CEREAL. Ingredients: Corn meal, malt syrup, di-calcium phosphate, yeast, salt, sodium iron pyrophosphate, niacinamide, thiamine hydrochloride, and riboflavin.

Analysis (submitted by manufacturer).—Total solids 93.8%, moisture 6.2%, ash 4.5%, fat (acid hydrolysis) 3.0%, protein 6.5%, crude fiber 1.0%, and carbohydrates 78.8%.

Vitamins and Minerals	Mg. per 100 Gm.
Thiamine	1.6
Riboflavin	1.5
Nicotinic acid	3.5
Calcium	800
Phosphorus	900
Iron	50

Calories.—3.6 per gram; 104 per ounce.

Use.—For use in the feeding of infants and young children.

Dairyland Cooperative Association, Juneau, Wisconsin.

PAR-EX. Ingredients: Evaporated milk, vitamin D_3, Dyne (irradiated 7-dehydrocholesterol), thiamine, U.S.P., nicotinic acid, U.S.P., ascorbic acid, U.S.P., ferrous sulfate, U.S.P., copper sulfate, U.S.P., manganese sulfate, U.S.P., di-sodium phosphate less than 0.1%.

Analysis (submitted by manufacturer).—Total solids 26.0%, moisture 74.0%, ash 0.7%, fat 7.9%, protein (N × 6.35) 7.0%, carbohydrate 9.4%.

Vitamins and Minerals	Per Can (12 Fluidounces)
Vitamin A	1,500 U.S.P. Units
Thiamine	0.5 mg.
Riboflavin	1.0 mg.
Ascorbic acid	50.0 mg.
Vitamin D_3 (irradiated 7-dehydrocholesterol)	800 U.S.P. Units
Nicotinic acid	5.0 mg.
Calcium	800 mg.
Phosphorus	800 mg.
Iron	10.0 mg.
Manganese	1.0 mg.
Copper	1.0 mg.

Calories.—569 per can, 43 per fluidounce.

Use.—For use in infant feeding.

Libby, McNeill & Libby, Chicago.

LIBBY'S STRAINED AND HOMOGENIZED BANANA PUDDING. Ingredients: Banana puree, milk solids (non-fat), wheat starch, corn starch, sugar, vanilla and sufficient water for preparation.

Analysis (submitted by manufacturer).—Total solids 29.3%, moisture 70.7%, ash 0.75%, fat 0.077%, protein (N × 6.25) 3.07%, crude fiber 0.031%, carbohydrate other than crude fiber (by difference) 25.37%.

Vitamins and Minerals	Per 100 Gm.
Carotene	64 I.U.
Thiamine	0.028 mg.
Riboflavin	0.182 mg.
Ascorbic acid	1.20 mg.
Nicotinic acid	0.149 mg.
Calcium	114 mg.
Phosphorus	90 mg.
Iron	4.8 mg.

Calories.—1.14 per gram; 32.5 per ounce.

Use.—For use in the feeding of infants, young children, convalescents, and others requiring a soft diet.

Swift and Company, Chicago.

SWIFT'S DRIED EGG YOLKS FOR BABIES. Ingredients: Pasteurized, dried egg yolks.

Analysis (submitted by manufacturer).—Total solids 97.3%, moisture 2.7%, ash 3.5%, fat 59.9%, protein 33.1%, salt 0.1%, carbohydrate (by difference) 0.8%.

Vitamins and Minerals	Per 100 Gm.
Vitamin A	5,800 I.U.
Thiamine	0.4 mg.
Riboflavin	0.7 mg.
Nicotinic acid	0.4 mg.
Vitamin D	670 I.U.
Calcium	230 mg.
Phosphorus	870 mg.
Iron	8.3 mg.

Calories.—6.8 per gram; 193 per ounce.

Use.—For use in the feeding of infants, young children, and in special diets.

The following products, intended for use in low sodium and other therapeutic diets, have been accepted as conforming to the rules of the Council. Data regarding composition represent the best available information, which is based on submitted reports of analyses. The Council has requested continuing analytical studies, especially of the sodium content of products intended for use in low sodium diets, because of the natural variations in the composition of processed foods.

JAMES R. WILSON, M.D., *Secretary.*

S. S. Pierce Company, Boston.

OVERLAND BRAND DIETETIC PACK CUT GREEN BEANS consists of Tendergreen variety beans, packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 5.2%, moisture 94.8%, ash 0.23%, fat (ether extract) 0.11%, protein (N × 6.25) 0.88%, crude fiber 0.53%, carbohydrate 3.45%, and sodium 1.02 mg./100 gm.

Calories.—0.18 per gram; 5 per ounce.

Use.—For use in low calorie, low sodium, and other therapeutic diets.

OVERLAND BRAND DIETETIC PACK CUT WAX BEANS consists of Kinghorn variety wax beans, packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 4.6%, moisture 95.4%, ash 0.24%, fat (ether extract) 0.09%, protein (N × 6.25) 0.82%, crude fiber 0.54%, carbohydrate 2.91%, and sodium 0.80 mg./100 gm.

Calories.—0.15 per gram; 4 per ounce.

Use.—For use in low calorie, low sodium, and other therapeutic diets.

OVERLAND BRAND DIETETIC PACK DICED BEETS consists of Detroit dark red beets, packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 6.2%, moisture 93.8%, ash 0.44%, fat (ether extract) 0.05%, protein (N × 6.25) 0.76%, crude fiber 1.41%, carbohydrate 4.54%, and sodium 19.40 mg./100 gm.

Calories.—0.22 per gram; 6 per ounce.

Use.—For use in low calorie, low sodium, and other therapeutic diets.

OVERLAND BRAND DIETETIC PACK DICED CARROTS consists of Red Cored Chantenay carrots, packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 6.56%, moisture 93.44%, ash 0.48%, fat (ether extract) 0.06%, protein (N × 6.25) 0.78%, crude fiber 0.60%, carbohydrate 4.64%, and sodium 50.1 mg./100 gm.

Calories.—0.22 per gram; 6 per ounce.

Use.—For use in low calorie, low sodium, and other therapeutic diets.

OVERLAND BRAND DIETETIC PACK WHOLE KERNEL CORN consists of Tendermost variety corn, packed in water without any added salt

Analysis (submitted by distributor).—Total solids 17.4%, moisture 82.6%, ash 0.38%, fat (ether extract) 0.51%, protein (N × 6.25) 2.04%, crude fiber 0.33%, carbohydrate 14.14%, and sodium 1.2 mg./100 gm.

Calories.— 0.69 per gram; 20 per ounce.

Use.—For use in low sodium and other therapeutic diets.

OVERLAND BRAND DIETETIC PACK SWEET PEAS consists of peas (surprise variety), packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 11.7%, moisture 88.3%, ash 0.35%, fat (ether extract) 0.20%, protein (N × 6.25) 3.14%, crude fiber 1.34%, carbohydrate 6.67%, and sodium 8.58 mg./100 gm.

Calories.—0.41 per gram; 12 per ounce.

Use.—For use in low sodium and other therapeutic diets.

OVERLAND BRAND DIETETIC PACK SPINACH consists of Northland-Viking spinach, packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 5.7%, moisture 94.3%, ash 1.14%, fat (ether extract) 0.31%, protein (N × 6.25) 1.83%, crude fiber 0.60%, carbohydrate 1.82%, and sodium 9.10 mg./100 gm.

Calories.—0.17 per gram; 5 per ounce.

Use.—For use in low calorie, low sodium, and other therapeutic diets.

THE JOURNAL
OF THE AMERICAN MEDICAL ASSOCIATION
535 N. DEARBORN ST. . . . . CHICAGO 10, ILL.
Edited by . . . . . AUSTIN SMITH, M.D.
Associate Editor . . . . . JOHNSON F. HAMMOND, M.D.
Editor for Medical Literature Abstracts . GEORGE HALPERIN, M.D.

Subscription price . . Fifteen dollars per annum in advance
Cable Address . . . . . "Medic, Chicago"

FOLIC ACID DEFICIENCY IN MACROCYTIC ANEMIA OF PREGNANCY AND INFANCY

More than seven years have elapsed since folic acid was synthesized.[1] It has been established as a therapeutic agent. It produces a hemopoietic response in persons with pernicious anemia while not arresting the progress of the neurological disturbances, and may be life-saving in the treatment of the macrocytic anemia of pregnancy or the megaloblastic anemia of infancy. In some instances there is no substitute for folic acid therapy.[2]

Folic acid is a water soluble vitamin and is a member of the B complex. It is a yellow crystalline substance having the chemical formula of $C_{19}H_{19}N_7O_6$, and chemically it is termed pteroylglutamic acid. It exists in natural sources in several chemical forms. It is more abundant in green leaves, liver, and yeast than in any other foods.

Angulo and Spies [3] have shown that the diets eaten by many patients with sprue and by women with macrocytic anemia of pregnancy are low in folic acid content. It has been recommended that the macrocytic anemia of pregnancy can be prevented by treating the mother during pregnancy with substances like folic acid or foods containing adequate amounts of folic acid.[4]

Elsewhere in this issue, Spies, Dreizen, Parker, and Silberman [5] bring forward a great deal of evidence concerning folic acid deficiency in human beings. By administering folic acid to a mother with the macrocytic anemia of pregnancy who was nursing an infant with the megaloblastic anemia of infancy, these investigators observed that the mother and infant responded dramatically. The redness of the buccal mucosa in both mother and nursing infant faded, and each had a characteristic reticulocyte peak in the peripheral blood on the seventh day of therapy. Both the mother and infant gained strength as the red blood cells and hemoglobin increased. This demonstrates clearly the physiological relationship between the failure of hemopoiesis in the mother and failure of hemopoiesis in the nursing infant due to a deficiency of folic acid. These investigators also describe cases of pregnant women in whom this type of anemia developed while they were taking vitamin preparations that did not contain folic acid.

These observations demonstrate why Castle,[6] Wills,[7] Spies,[8] and others, after finding that certain patients did not respond to the administration of the purified antipernicious anemia liver extract, formerly recommended the use of crude fractions of liver and yeast. We now know that most potent antianemia liver extracts are rich in vitamin B_{12} but are almost devoid of folic acid.

The fact that liver extract, vitamin B_{12}, and folic acid each fail in the treatment of some types of macrocytic anemia was covered in an editorial in the Nov. 3, 1951, issue of THE JOURNAL. Nothing takes the place of diagnostic acumen and appreciation of the activity and specificity of the various vitamins. The fact that folic acid does not relieve acute neural degeneration is not a general indictment against it, for its value in some types of macrocytic anemia is beyond dispute. Likewise, the fact that vitamin B_{12} does not always relieve the macrocytic anemia of pregnancy and the megaloblastic anemia of infancy is no indictment against it. Vitamin B_{12}, or substances that act similarly, are essential for the integrity of the nervous system. Folic acid is essential in some types of macrocytic anemia; hence, both vitamin B_{12} and folic acid are essential for life.

SURGERY IN PATIENTS WITH CORTISONE-INDUCED ADRENAL INSUFFICIENCY

The cortex of the adrenal gland may partially atrophy during long-term treatment with cortisone. Should this happen, the natural supply of cortical steroids may not be sufficient at a time of acute stress, such as during and after anesthesia and operation, when steroids are essential to prevent shock and circulatory collapse. Anesthesia and operation are well tolerated when such patients are prepared preoperatively by the administration of cortisone.

Perhaps the most satisfactory schedule for preparing patients who are known to have adrenocortical suppression consists in the intramuscular administration of 100 to 200 mg. of cortisone per day for two or three days before operation and on the day of operation. A similar schedule also may be used for two or three days postoperatively, with a gradual reduction of the dose until it reaches the previous optimal figure.

On the other hand, there are instances of emergency in which a patient requires anesthesia and immediate operation. In such cases no such schedule of treatment

1. Angier, R. B., and others: Synthesis of a Compound Identical with the L. Casei Factor Isolated from Liver, Science **102**: 227, 1945.

2. Spies, T. D., and others: Therapeutic Indications for Vitamins in Mixtures: Principles in the Treatment of Nutritive Failure, Postgrad. Med. **10**: 269, 1951.

3. Angulo, J. J., and Spies, T. D.: The Determination of the Folic Acid Content of Foods Usually Consumed by Patients with Tropical Sprue, Am. J. Trop. Med. **27**: 317, 1947.

4. Spies, T. D.: Observations on the Macrocytic Anemia Associated with Pregnancy, Surg., Gynec. & Obst. **89**: 76, 1949.

5. Spies, T. D.; Dreizen, S.; Parker, G. S., and Silberman, D. J.: Recent Observations in the Detection and Treatment of Nutritive Failure in Children, read before the Section on Pediatrics at the One-Hundredth Annual Session of the American Medical Association, Atlantic City, N. J., June 14, 1951, J. A. M. A. **148**: 1376 (April 19) 1952.

is possible; hence it is imperative to ask such a patient or someone who can speak for him whether he has been receiving cortisone or corticotropin (ACTH). If so, use of about 30 to 50 cc. of rapidly acting aqueous adrenocortical extract injected intravenously and repeated as needed, in addition to resumption of cortisone or corticotropin therapy, is imperative to maintain the unprepared patient safely during the period of stress so that he may be assured thereby of the support necessary for survival.

This matter is sufficiently important to justify a warning to internists, surgeons, and anesthesiologists to be certain that cortisone, corticotropin, and aqueous adrenocortical extract is available for the immediate treatment of the patient undergoing anesthesia and operation whose stress response mechanism may be impaired due to prior treatment with cortisone or corticotropin. It is imperative that such a patient receive proper treatment preoperatively, during the operation, and postoperatively. If this is done, it is believed that lives will be saved.

PROCAINE IN ATHLETIC INJURIES

The use of procaine hydrochloride and related anesthetic agents by injection locally to relax muscle spasm and improve circulation to the part by allowing motion free of pain has been advocated by Bettmann,[1] Grieve,[2] Murphy,[3] and others. Although some authors do not employ immobilization of any kind or advise rest in conjunction with the treatment of injury of the lower extremities, their reports generally lack long follow-up periods and careful evaluation of the late results of this method of treatment. Despite the fact that ordinary physical activity may be encouraged and cases of unusually heavy work being carried out are described, none directly advises active participation in athletic contests immediately after treatment. Quigley [4] states, "Novocaine has no place in the treatment of ligament sprains in athletes. Under its influence a minor sprain might easily become major."

In spite of the lack of good precedent to justify the practice, the use and abuse of local anesthetic agents to enable amateur and professional athletes to continue active sports participation in the face of serious injury is apparently becoming more widespread. Hardly a month passes that even the casual reader of the sports pages does not find a reference to the use of injection of "pain-killing drugs" in connection with some prominent athletic figure. Many will remember the case of the baseball catcher who finished the last two games of the World Series a few years ago limping on an ankle which was heavily taped and injected with procaine hydrochloride, only to discover when a roentgenogram was taken that a serious fracture had been missed and, therefore, neglected.

Last fall, just before the World Series, an article about another catcher appeared under an Associated Press dateline from which the following excerpt is taken: "Campanella returned after taking his licks. 'That novocaine didn't do no good yesterday,' he said. 'Don't think this stuff will do any good neither. It hurts too deep.' 'How about it? Do you want to play?' Dressen finally asked. Campanella shrugged. 'It's all right with me,' he said and hobbled away." Only the day before in an article by the same writer, the following quotation was given from the manager of another major league team: "Do you know Jansen has had a bad back for the last month? Has to have shots to kill the pain before he pitches, but when I tell him he is pitching, he just says, 'Where's the ball?' 'You can't beat spirit like that.' " One month later the repeated use of procaine for a prominent pitcher during the season of 1945 was revealed by his manager and confirmed by the physician who had given the injections. The latter was quoted also as saying that he had used procaine (novocaine®) "many times" on players of the Detroit Tigers baseball team, the Detroit Lions professional football team, and the University of Detroit football team. "There is no dope situation involved," he said. "The drug is merely used as an aid for healing." This aroused even a seasoned sports columnist to the following comment: "Does Dr. ———, the University of Detroit trainer, who recently defended his use of novocaine on pitcher Hal Newhouser's sore arm in 1945 to kill the pain, on the grounds that it is a healing aid and is used regularly in training rooms, know that medical books describe it as an anesthetic with less toxicity than cocaine, and, far from having healing properties, did in the case of Newhouser, cause him to aggravate his arm injury by pitching while the pain was numbed by the drug. Incidentally, if it is illegal to use drugs in a contest involving dogs or horses, why shouldn't the ban be extended to include athletes for their own protection?"

The use of adhesive tape and procaine to enable football, basketball, and hockey players, as well as track stars, to continue to participate during the competitive season is common knowledge. Its use is so general that there is reason to believe that trainers, physiotherapists, and other unauthorized persons are actually employing injections of local anesthetic drugs without proper medical supervision. The various pressures, financial or otherwise, that have made winning more important than playing have been aired thoroughly during the last year. They have been responsible for allowing many young men to risk serious, permanent injury for pay or prestige. Such indiscriminate use of procaine should not be tolerated by those interested in the welfare of athletes.

---

6. Castle, W. B., and Minot, G. R.: Pathological Physiology and Clinical Description of the Anemias, London, Oxford University Press, 1936.

7. Wills, L., and Mehta, M. M.: Studies in "Pernicious Anemia" of Pregnancy: Preliminary Report, Indian J. M. Res. **17**: 777, 1930.

8. Spies, T. D., and Butt, H. R.: Vitamins and Avitaminosis, in Duncan, G. G.: Diseases of Metabolism, ed. 3, Philadelphia, W. B. Saunders Company, 1952, p. 401.

1. Bettmann, E. H.: Prolonged Therapeutic Action of Intracaine in Painful Musculoskeletal Disorders, New York State J. Med. **47**: 2193 (Oct. 15) 1947.

2. Grieve, J.: Procaine in the Treatment of Minor Injuries, Lancet **1**: 344 (March 15) 1941.

3. Murphy, F. C., and Postlethwait, R. W.: Novocain Injection for Minor Injuries in Military Service, Surg., Gynec. & Obst. **77**: 397 (Oct.) 1943.

4. Quigley, T. B.: The Management of Knee Injuries Incurred in College Football, Surg., Gynec. & Obst. **87**: 569 (Nov.) 1948.

ORGANIZATION SECTION

REPORT OF BOARD OF TRUSTEES

The following report of the Board of Trustees will be presented to the House of Delegates of the American Medical Association at its meeting to be held in Chicago in June:

FINANCIAL STATEMENT

The official reports of the Treasurer and of the Association's auditors are appended as a part of this report.

In 1951 the $25.00 annual membership dues of the American Medical Association included a subscription to THE JOURNAL, and a Member Fellow had the privilege of substituting another scientific publication of the Association. In the prior year the payment of membership dues did not include a subscription.

The constituent state and territorial medical associations reported collections of membership dues in 1951 of $2,777,539. An estimate at the close of the year of additional collections of 1951 dues, amounting to $122,461, appears justified at the time of this writing, March 15, 1952. Realized and estimated collections together total $2,900,000, equivalent to 116,000 member payments. Fifteen dollars of each member payment is allocated to the income of the periodical chosen by the member, and after this allocation the income credited to 1951 membership dues amounted to $1,159,683. This amount is identified in the Statement of Income in the financial statements.

Additional collections of 1950 dues were reported in 1951 in the amount of $332,724. A total of $228,965 was paid in 1951 Fellowship dues, equivalent to 45,793 Fellowship payments. The payment of Fellowship dues was discontinued by the Board of Trustees with the year 1952, and income from this source will not be recurring.

Association investments produced income of $167,762, an approximate average return of 2½%. A portion of Association funds is invested in short-term government securities yielding low interest rates. A general classification of Association securities may be found in the Balance Sheet.

Miscellaneous receipts and other income amount to $68,670. The total of the foregoing items, $1,957,804, represents income in 1951 exclusive of income from the periodical publications.

Subscription income from THE JOURNAL, the monthly periodical publications, and *Today's Health*, amount to $3,094,797. Advertising income from the same sources amounts to $2,686,719. Subscription and advertising income together total $5,781,516. Printing and other costs incident to the publication of the periodicals were $3,613,254, making a net income of $2,168,262. Total income from all sources amounted to $4,126,066.

The expenses of the Councils, Bureaus, and related activities totaled $2,243,880. This compares with $1,838,382 in 1950. The increase represents in part higher operational costs, but it also reflects an expansion in functions and service. This is particularly so in the Department of Public Relations, which in 1951 expended $209,212 compared with $102,116 in 1950, and in the Council on Medical Education and Hospitals, which expended $212,018 in 1951 and $177,767 in 1950. The Bureau of Health Education, owing to higher radio costs, and some experimentation with television, spent $208,788 in 1951 compared with $150,578 in 1950, and the 1951 expenditures of the Bureau of Medical Economic Research amounted to $180,329 compared with $140,311 in 1950. The expenditure of each Council, Bureau, and related activity for the year 1951, together with a consolidated classification of the expenses, appears in the auditor's report.

Employee past service annuities and retirement pay of $135,126 and legal fees and sundry, $7,042, amount to $142,168.

Economies have been introduced in the printing of the Quarterly Cumulative Index Medicus by adoption of the offset printing method. The loss incurred in publishing and printing the Index amounted to $49,768 in 1951 and $71,878 in 1950, a reduction in loss of $22,110.

Other items of expense include a loss from the sale of pamphlets, reprints, and books, cash discounts allowed over those received from vendors, and depreciation on office equipment.

The Annual and Clinical Sessions together resulted in a net loss of $79,738 in 1951. The net loss of the two sessions amounted to $71,297 in 1950.

During 1951, $529,515 was expended on the National Education Campaign. This compares with an amount of $2,593,386 expended in 1950, which was paid from American Medical Association dues collections of that year.

An amount of $500,000 was appropriated from 1951 income in support of the American Medical Education Foundation, and a similar sum was appropriated from 1950 income.

At the close of 1951 there were 871 persons employed in the offices and in the printing plant of the Association, which compares with 841 persons the year before. Salaries and wages paid in 1951 amounted to $3,329,109, as compared with $3,049,247 in 1950. A total of 4,580 tons of paper was used in printing Association periodical publications in 1951, at a cost of $751,800, as compared with 3,814 tons used in 1950 at a cost of $602,216. The action of the House of Delegates in providing a subscription to THE JOURNAL for all dues paying members accounts for the greater part of this increase in paper consumption. Social Security taxes paid in 1951 amounted to $49,900; in 1950, $43,485.

Capital expenditures made during 1951 in the headquarters building property amounted to $19,483, and expenditures for new printing equipment, office furniture, and laboratory equipment amounted to $65,901.

The Board of Trustees approved a project in the closing months of the year to mechanize the Membership, Fellowship, and Subscription Department records. The installation, which is now nearing completion, will expedite the operations of the Department and enhance its service to the membership at large and to the constituent associations.

The Association, through the exercise of rights acquired on its current holdings of common stocks, added only $4,232 to its investment in common stocks during the year. At the end of 1951, marketable securities in the portfolio of the Association at cost prices totaled $5,049,334, exclusive of $1,493,529 in securities held in the American Medical Association Research Fund; together $6,542,863. This compares with securities totaling $6,240,252 at the end of 1950. Marketable securities in the amount of $5,049,334 represent investments of:

General Fund	$ 919,334
Association Reserve Fund	350,000
Building Reserve Fund	450,000
Retirement Reserve Fund	675,000
Equipment Modernization Fund	700,000
Depreciation Reserve Fund	1,455,000
AMEF Reserve Fund	500,000
	$5,049,334

TREASURER'S REPORT

REPORT OF THE TREASURER OF THE AMERICAN MEDICAL ASSOCIATION FOR THE YEAR ENDED DEC. 31, 1951

Investments (at cost) as at Jan. 1, 1951		$4,750,148.48	
Bonds purchased (at cost)	$3,069,857.73		
Common stocks purchased (at cost)	4,132.00	3,073,989.73	
		$8,424,138.21	
Less:			
Bonds called, matured, or sold		3,374,804.38	
Investments as at Dec. 31, 1951			$5,049,333.83
Balance held for investment Jan. 1, 1951	$ 37,752.48		
Interest and dividends received in 1951	174,709.74		
	$ 212,462.22		
Transferred to general fund	176,826.71		
Uninvested funds as at Dec. 31, 1951			85,635.51
Invested and uninvested funds as at Dec. 31, 1951			$5,084,969.34

AMERICAN MEDICAL ASSOCIATION RESEARCH FUND

Investments (at cost) as at Jan. 1, 1951	$1,490,103.13	
Bonds purchased (at cost)	13,528.76	
	$1,503,631.89	

Less: Bonds sold	10,103.13	
Investments as at Dec. 31, 1951		$1,493,528.76
Uninvested funds as at Jan. 1, 1951	3,996.87	
Less: Expended for purchase of securities	3,450.80	
Uninvested funds as at Dec. 31, 1951		546.07
Invested and uninvested funds as at Dec. 31, 1951		$1,494,074.83
Interest received on investments and transferred to general fund to apply on research expenditures	$ 36,826.71	

DAVIS MEMORIAL FUND

Balance in funds Jan. 1, 1951	$ 8,412.08	
Interest earned on bank balance in 1951	116.06	
Funds on deposit at Dec. 31, 1951		$ 8,528.14

JOSIAH J. MOORE, Treasurer.

AUDITOR'S REPORT

The Board of Trustees
American Medical Association

March 31, 1952

We have examined the balance sheet of the American Medical Association as of Dec. 31, 1951, and the related statement of income for the year then ended. Our examination was made in accordance with generally accepted auditing standards, and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances.

In our opinion, the accompanying balance sheet and statement of income present fairly the financial position of American Medical Association at Dec. 31, 1951, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles which, except for the change (of which we approve) in the method of recording dues income as set forth in the note to the statement of income, have been applied on a basis consistent with that of the preceding year.

PEAT, MARWICK, MITCHELL & CO.

STATEMENT OF INCOME

FOR THE YEAR ENDED DEC. 31, 1951

Income:		
Membership dues:		
1951 dues less $1,739,525 (amount of allocation to periodical subscription income based on $15.00 for each membership payment)		$1,159,683
1950 dues		332,724
		1,492,407
Fellowship dues		228,965
Income from investments		167,762
Miscellaneous receipts and other income		68,670
		1,957,804
Publications (periodicals):		
Subscriptions, including $1,739,525 allocated from 1951 membership dues	$3,094,797	
Advertising	2,686,719	
	5,781,516	
Less printing and other costs (Schedule 1)	3,613,254	2,168,262
		4,126,066
Expenses:		
Councils, bureaus, and related activities (Schedule 2)	2,243,880	
Employees' past service annuities and retirements	135,126	
Accumulated expenses and provision for completion costs of publishing Quarterly Cumulative Index in excess of accumulated income therefrom	49,768	
Costs and other expenses of printing and procuring books, pamphlets, and reprints in excess of income therefrom	71,162	
Annual and interim sessions—net	79,738	
Cash discounts—net	37,701	
Depreciation and taxes unallocated	28,330	
Legal and sundry	7,042	
	2,652,747	
Expenditures for National Education Program	529,515	
Appropriated for The American Medical Education Foundation	500,000	3,682,262
Income in excess of expenses		$ 443,804

Note—During the year 1951, the Association received 1950 membership dues amounting to $332,724. These dues were recorded as income when received. The Association has recorded 1951 membership dues on an accrual basis and the accounts include $122,461 of 1951 membership dues receivable at Dec. 31, 1951.

AMERICAN MEDICAL ASSOCIATION—Balance Sheet—Dec. 31, 1951

Assets

Cash			$ 89,715
Accounts receivable:			
Advertising		$ 253,978	
Membership dues		122,461	
Directory report service—19th edition		38,346	
Other		81,533	496,318
Interest accrued on investments			31,539
Inventories of materials, supplies, work in progress, and publications, at cost			301,433
Expenditures on publication in progress			94,143
Prepaid expenses, deposits, and advances:			
Advances to cover commitments of the National Education Program		72,400	
Other deposits and advances		41,756	
Insurance and other prepaid expenses		29,118	143,274
Marketable securities at cost (value based on market quotations, $4,950,067):			
United States government securities		2,818,321	
Railroad, municipal, public utility and industrial bonds		1,802,809	
Common stocks		428,204	5,049,334
Representing investments of:			
The American Medical Education Foundation Fund	$ 500,000		
General fund	919,334		
Depreciation reserve fund	1,455,000		
Association reserve fund	350,000		
Retirement reserve fund	675,000		
Building reserve fund	450,000		
Equipment modernization reserve fund	700,000		
American Medical Association Research Fund:			
United States government securities at cost (value based on market quotations, $1,433,234)		1,493,529	
Cash		546	1,494,075
Property, plant, and equipment, at cost:			
Land		328,774	
Building	2,126,592		
Machinery and printing equipment	691,082		
Office and laboratory equipment	442,082		
	3,259,756		
Less reserve for depreciation	1,453,216	1,806,540	2,135,314
			$9,835,145

Liabilities

Accounts payable and accrued expenses:			
State Journal Advertising Bureau			$ 46,726
Other accounts payable			158,575
Accrued payroll			52,500
Accrued taxes			68,061
			325,862
Provision for payment to The American Medical Education Foundation			500,000
Provision for completion costs of publications to be issued			183,700
Deferred credits:			
Unexpired subscriptions to publications		$ 380,254	
Income from 19th edition directory report service less accumulated costs applicable to directory		48,825	
Other		15,602	444,681
American Medical Association Research Fund reserve			1,494,100
Other reserves:			
Association		350,000	
Retirement		675,000	
Building		450,000	
Equipment modernization		700,000	2,175,000
Capital account:			
Balance at Dec. 31, 1950		4,267,998	
Excess of income over expenses for year ended December 31, 1951		443,804	4,711,802
			$9,835,145

PUBLICATIONS (PERIODICALS) PRINTING AND OTHER COSTS

FOR THE YEAR ENDED DEC. 31, 1951

Salaries and wages	$1,306,463
Paper and ink	888,129
Illustrations	101,567
Contract printing	149,051
Postage and cartage	205,375
Editorial (excluding wages)	134,250
Advertising sales and services (excluding wages)	96,467
Circulation (excluding wages)	306,705
Other costs—including allocation of building and plant maintenance, depreciation, taxes, and employee welfare tax	425,247
	$3,613,254

EXPENSES OF COUNCILS, BUREAUS, AND RELATED ACTIVITIES

FOR THE YEAR ENDED DEC. 31, 1951

Salaries and wages	$1,027,187
Department printing	107,989
Office supplies and equipment maintenance	50,810
Postage, express, and freight	58,830
Telephone and telegraph	23,886
Memberships, books, and periodicals	25,942
Legislative services	4,206
Statistical equipment rental	20,005
Membership records and reimbursement to state societies for dues collections	64,293
Biographical records	80,576
Staff and officers' travel	105,258
Inspection of hospitals and medical schools	18,518
General administrative expense, including allocation of headquarters maintenance, employee insurance, and taxes	207,173
Educational exhibits, motion pictures, and literature	94,100
Radio and television	89,543
Trustee meetings	14,463
Committee conferences	195,794
Grants, tests, and surveys	16,238
Miscellaneous	39,069
	$2,243,880

Note—The above expenses are allocated to the following councils, bureaus, and related activities:

Association	$ 143,234
Secretary's Office	83,538
Department of Public Relations	209,212
Washington Office of A. M. A.	180,483
Biographical Records	80,576
Membership Records	64,293
A. M. A. Laboratory	68,943
Microbiologic Laboratory	11,957
Council on Medical Education and Hospitals	212,018
Council on Medical Service	100,338
Council on Pharmacy and Chemistry	113,290
Council on Physical Medicine and Rehabilitation	56,946
Council on Foods and Nutrition	41,336
Council on Industrial Health	67,887
Council on National Emergency Medical Service	39,851
Council on Rural Health	31,546
A. M. A. Library	54,840
Bureau of Exhibits	65,320
Bureau of Health Education	208,788
Bureau of Investigation	21,214
Bureau of Legal Medicine and Legislation	51,484
Bureau of Medical Economic Research	180,329
Committee on Cosmetics	15,301
Committee on Research	39,556
Commission on Chronic Illness	10,000
Survey of Medical Education	64,054
The American Medical Education Foundation	14,641
Student American Medical Association	12,905
	$2,243,880

THE GOVERNMENT AS THE INSURED OF A PRIVATE INSURER

At the Clinical Session of the American Medical Association in December, 1951, a special committee, under the chairmanship of Dr. Elmer L. Henderson, was appointed by the Board of Trustees to study and report with respect to the following aspects of federal medical services:

(*a*) medical and hospital benefits for veterans with non-service-connected disabilities; (*b*) medical and hospital benefits for dependents of service personnel, and (*c*) the transfer of seriously disabled service personnel from service hospitals to Veterans Administration installations.

It is intended that a very thorough and exhaustive study be made of the laws and their legislative history as well as administrative regulations and procedures dealing with these subjects. It is also planned to compile pertinent information reflecting costs, trends, and future plans in these three areas. The results of the study will be used as the basis for policy recommendations to the Board of Trustees and the House of Delegates of the Association.

In considering the possibility of furnishing medical care for certain federal beneficiaries by means of prepaid health insurance, it has been suggested that the premiums under such a program be paid by the federal government. It is believed that the following memorandum, addressed to Dr. George F. Lull from Frank G. Dickinson, Ph.D., Director of the Bureau of Medical Economic Research, which discusses this possibility, will be of interest.

The activities of Dr. Henderson's Committee and the contents of Dr. Dickinson's memorandum have been discussed briefly in the Secretary's Letter (a letter from George F. Lull, Secretary, to state and county medical associations). As indicated therein by Dr. Lull, any suggestions or comments in regard to either subject should be addressed to Mr. C. Joseph Stetler, Secretary of the Committee on Legislation, who has been assigned the staff responsibilities in connection with this work.

TO: George F. Lull, M.D.
FROM: Frank G. Dickinson, Ph.D.
SUBJECT: Should the Federal Government Be Insured by a Private Insurer?

1. Incident to the actions of the House of Delegates at Los Angeles, Dec. 3-6, 1951, (*J. A. M. A.*, Dec. 22, 1951, Novy, pages 1697-1698, McKeown, page 1698, Appel, pages 1700-1701, Wall, page 1701, Hamilton, page 1701, and Robbins, page 1701), you have asked me to prepare a statement on the general question stated above. You suggested that I omit discussion of the purchase of insurance by local and state governments because the question relates to the federal government only. The extent of the insurance operations of federal and state governments as insurers, not as insureds, has been outlined in a booklet, "Government Insurance in the United States," by Frank Lang.

2. The federal government has never, to my knowledge, purchased insurance from a private company or firm. A review of insurance history might possibly uncover a few isolated and unimportant instances. During the December, 1951, meeting of the American Association of University Teachers of Insurance, of which I was president for two years, I asked the following authorities for references to instances: (1) Prof. Ralph H. Blanchard, Columbia University, (2) Prof. Harry J. Loman, University of Pennsylvania, (3) Prof. John H. Magee, formerly University of Maine, and (4) Prof. David McCahan, University of Pennsylvania. (Blanchard is editor of the McGraw-Hill Insurance Series, Loman is dean of the American Institute for Property and Liability Underwriters, Magee is the author of several widely used insurance books, and McCahan is executive vice-president of the College of Life Underwriters.)

3. Each of these four authorities has told me that, to the best of his knowledge, the federal government has never purchased insurance from a private insurance company. It has and does often require insurance of persons and firms that sell goods or services to it, or persons who apply for loans. The federal government has probably never been an insured of a private insurer.

4. Should the federal government by the payment of premiums become insured by a private insurance company? An examination of the fundamental principles of insurance will indicate clearly that the answer must be no. First consider a large business firm and its need for insurance. Suppose that the ABC Distributing Company owned 10,000 store buildings of approximately the same size scattered throughout the length and breadth of the United States. Suppose further that evidence had been accumulated that indicated that one-twentieth of one per cent of these buildings would, on the average, be completely destroyed by fire during any one calendar year and that one-third of one per cent would be damaged by fire. Should this company purchase fire insurance on all of these buildings? It would be quite unnecessary. This company might—indeed, some companies do—employ an insurance organization to investigate the claims, the causes of the fire, particularly the physical and human causes, and take care of any legal matters incident thereto. For this service the insurance company is paid a fee of, say, 10%, but the operation is a service and not an insurance operation. The insurance company in performing this service for the ABC Distributing Company would not have guaranteed the loss ratio, would not have guaranteed, as it does its insureds, that for the premium paid the entire losses covered by the contract would be paid no matter how great or how small the losses might be during the

policy period, usually one to three years. So much for insurance principles in relation to a large business firm.

5. Now consider the application of insurance principles to the sovereign power. There is no sound reason in insurance history or in the nature of insurance for the federal government to become an insured of a private insurance company. In the illustration in 4 above I refer to a very uncommon service; property insurance companies do occasionally contract to render this type of service to self-insured business firms with sufficient spread of risk to warrant self insurance. (The propriety of the federal government paying the gross premiums—net loss plus overhead, including some financial return on the risk capital—is beyond the scope of these remarks.) The federal government from the standpoint of insurance is 155 million persons, the broadest possible base for the distribution of risk. It is larger than thousands of ABC Distributing Companies. Indeed, it is larger than all insurance companies combined, because it is the sovereign power. The federal government is all of us. Subject only to the discretion of Congress and of the President and the requirements of uniformity imposed by the Constitution as interpreted by the courts, the federal government has a 100% lien on your future income and mine. The federal government is truly the sovereign power. Its borrowing power is not limited by the federal Constitution. The federal government does not and should not buy fire insurance, marine insurance, automobile insurance, bridge insurance, or workman's compensation insurance from private carriers, and, in the case in point, the federal government should not buy accident and health insurance from Blue Cross and Blue Shield Plans or insurance underwriters. The very notion of the federal government as the sovereign power eliminates it from any rational consideration of being made an insured of an insurance company. The only responsible federal official who has recently suggested that the federal government become an insured of a private company is Oscar Ewing, according to the Washington News of THE JOURNAL (147:14 [Dec. 29] 1951); "Federal Security Agency officials who are attempting to work out a program for hospitalization of the aged still are undecided whether to use voluntary insurance plans, with the federal government paying the premium . . . Mr. Ewing has expressed an interest in extending coverage through voluntary insurance plans and has conferred with officials of some of the insurance organizations." If Mr. Ewing should have this legislation introduced and it should be enacted into law, it would be, to my knowledge, the first instance of its kind. Perhaps when and if Mr. Ewing comes to understand why no agency of the federal government should be insured by a private agency, he will drop the matter.

6. In the resolutions cited in 1 above and in other discussions, I have gained the impression that the distinction between prepayment and postpayment in relation to insurance principles is not clear. There are many instances of premiums being adjusted at the end of some experience period. With certain exceptions there is a guarantee as to the maximum amount of the additional premium that will be determined at the end of the policy period. In some of our mutual companies, for example, the additional premium or assessment cannot exceed the amount of the original premium paid in advance. Insurance premiums are usually paid in their entirety at the beginning of the premium period. Insurance is usually, therefore, a prepayment and not a postpayment proposition.

7. It has been suggested that Blue Cross and Blue Shield plans and private insurance companies might enter into contracts with the federal government to service the claims of veterans with non-service-connected disabilities and the dependents of servicemen. Presumably, the "insurers" would investigate the claims, pay the physician, the dentist, the hospital, the druggist, etc., report these amounts monthly or quarterly to the proper official in Washington, and, after the disbursements were audited, the insurer would be reimbursed and paid some fee for his services, say 10%. This kind of postpayment for expenditures made on behalf of the client bears no resemblance to insurance and lies completely outside the scope of insurance as that subject has developed over the past three to five centuries. Indeed, the federal agency could select some other kind of company or association to do this same work at the local level. Presumably, the Blue Cross and Blue Shield plans and the private insurance companies would be selected solely because they could render the best service or because they could afford to perform this investigating and paying function at the lowest possible cost, but it could be done by banks, personal finance companies, or other business institutions.

8. Finally, would a private insurance company ever want to have the federal government as one of its insureds? Certainly the private firm could expect to have the (federal agency) insured dictate the terms of the contract, the premium rates, and the claim adjustments. When the Social Security Act of 1935 was before Congress there was a considerable amount of comment in the press and in Washington about the desirability of the federal government reinsuring the social insurance risks with private insurance companies. The companies themselves said: "No, thank you. We do not want the kind of business that would enable the federal government to dictate the terms of the annuities for persons covered by social security, because that would ultimately mean complete dictation of the contracts for all of our policyholders." The extent of this hazard for a private insurance company, Blue Cross plan, or Blue Shield plan, would depend in some measure on whether the amount of premium derived from a group contract insuring an agency of the federal government comprised a small or a large percentage of the total premium income of the private insurer. It seems likely that very few experienced insurance executives would care to take on the federal government as one of their company's insureds.

9. *Summary.*—(*a*) There is nothing in the history, theory, and principles of insurance to warrant the federal government becoming an insured of a private insurance company. It would make the sovereign power of government subservient to a private business institution. It would be contrary to public policy. (*b*) The Congress and the President would unquestionably reject any proposal that both violates the fundamental rules of insurance and seeks to invert the inherent relationship between the sovereign power and a private citizen, association, or firm. (*c*) Finally, the private insurer would certainly face the inevitable danger of absorption and control by its insured—an agency of the federal government.

FEDERAL MEDICAL LEGISLATION

Nurse Cadets for Military Forces

Representative Cole of New York introduced H. R. 7160, which would provide for the training of candidates for appointment as nurses in the military services. This bill would establish an officer candidate training program for nurses to meet military requirements. Appointments would be made to the regular armed services or to their reserve components. Candidates would be trained at institutions prescribed by secretaries of the Army, Navy, and Air Force. During the course of training all expenses would be paid for the trainee, including tuition, student uniforms, fees, books, school health programs, and laboratory expenses. A subsistence allowance of $50 per month would be paid trainees. Candidates would be required to agree to serve for one year of active duty for each year of subsidized training. At the option of the military services, candidates could be released or required to begin obligated service any time during the training period. On satisfactory completion of training, candidates would be eligible for appointments as ensigns or second lieutenants in the regular or reserve components of the military services.

Transfer of Indian Hospitals

H. R. 7232 was introduced by Representative Patten of Arizona. This bill proposes to transfer the administration of health services for Indians and the operation of Indian hospitals to the Public Health Service. It is identical with S. 2780 and H. R. 6908, previously reported.

Medical Benefits to Veterans' Dependents

A bill to grant free outpatient medical and dental treatment to certain children of deceased veterans was introduced by Representative O'Neill of Pennsylvania. This bill, H. R. 7320, would authorize the Administrator of the Veterans Administration to

---

The summary of federal legislation was prepared by the Washington Office of the American Medical Association and the summary of state legislation by the Bureau of Legal Medicine and Legislation.

furnish "necessary free out-patient medical and dental treatment to the child or children" of veterans who die or who have died in active military service as a result of disability incurred in or aggravated by such service in the line of duty. The Administrator would provide such treatment in the same manner and to the same extent as treatment furnished to veterans for service-connected disease or injury. The term "child" includes children up to age 18 and under certain circumstances to age 21.

Veterans Presumption of Service Connection for Nonpulmonary Tuberculosis

Representative Rankin of Mississippi introduced H. R. 7347, which would amend the veterans' regulations to establish for certain persons who served in the armed forces a further presumption of service connection for nonpulmonary forms of active tuberculosis. This bill is similar to a previously reported bill, H. R. 7022, and would amend Veterans' Administration regulations to establish for persons who served in the armed forces during wartime a three-year presumption of service connection for nonpulmonary tuberculosis. The present regulations establish a one year presumption of service connection for nonpulmonary tuberculosis for World War II veterans. This bill was introduced at the request of the American Legion.

STATE MEDICAL LEGISLATION

Arizona

Bill Enacted.—S. 4, has become Ch. 57 of the Laws of 1952. It provides certain amendments to the law relating to narcotic drugs including, among other things, a definition of the word **"physician" to mean a person licensed to practice medicine, or osteopathy,** or otherwise licensed to treat sick and injured human beings, and to use narcotic drugs in connection with such treatment.

California

Bills Introduced.—S. R. 34, proposes the creation of an interim commission to ascertain, study and analyze **all facts relating to all state hospitals and veterans' institutions** whatever their nature.

Kentucky

Bills Enacted.—H. R. 46, became Law without approval, March 26, 1952. It directs the legislative research commission in cooperation with the University of Kentucky to make a careful and impartial study of the desirability and steps necessary for the **establishment of a state supported medical school** of the University of Kentucky. H. 172, became Law without approval, March 24, 1952. It authorizes the Department of Health to establish and maintain services and facilities, including **hospital schools,** for the care and education for physically handicapped persons and to provide in connection therewith medical and nursing care. H. 279, was approved March 18, 1952. It repeals and reenacts the law **relating to the licensure of chiropodists** and defines chiropody to mean the diagnosis, and the local, medical, mechanical, and surgical treatment, of ailments of the human foot, and massage in connection therewith, except amputation of the foot or toes, or the use of anesthetics other than local, or the use of drugs or medicines other than local. S. 100, was approved March 20, 1952. It creates an **alcohol study commission** to make a comprehensive study on the problem of alcoholism, the existing methods and facilities in the state for treatment, the present program for public education on alcoholism, and the present status of the laws of the state in regard to alcoholism. S. 166, was approved March 25, 1952. It provides, among other things, that **no person shall be denied admission to any hospital** or clinic operated by any city-county board of health on account of his financial condition nor shall the administration of emergency treatment to such person be delayed on account thereof. S. 238, was approved March 24, 1952. It amends the workmen's compensation act by providing that employees and employers shall furnish to the opposite party **copies of any medical examinations** made of the employee either by a physician chosen by the employer or by the employee's own physician.

Maryland

Bills Enacted.—S. 5, has become Ch. 17 of the Laws of 1952. It includes isonipecaine, amidone, and dromoran within the definition of **"narcotic drugs,"** together with certain synthetic drugs having common habit-forming qualities and effects of habituation. S. 19, has become Ch. 19 of the Laws of 1952. It amends the law relating to **rabies** by making it the duty of any person having a knowledge of the fact that a person has been bitten by a dog having or suspected of having rabies to report the fact to the county health officer who shall see that such dog is confined for such period of time as the health officer may direct.

Massachusetts

Bill Introduced.—H. 2234, proposes to authorize the committee on labor and industries to make an investigation and **study relative to medical services under the workmen's compensation act;** controlling the disclosure of information contained in the employee's medical records; furnishing of copies of medical records, reports of hospitals, clinics, and physicians; and other subjects.

Michigan

Bill Introduced.—S. C. R. 43, proposes a commission of an interim commission to study the **mental health program** in the state.

Mississippi

Bills Introduced.—H. 951 and H. 979 propose an appropriation to defray the expenses of the state board of health and to be used, among other purposes, for the **inspecting of private hospitals** in the state which receive money from the state for charity patients with a view to approving or disapproving such hospitals for the reception and treatment of charity patients. H. 955, proposes an appropriation for defraying the expenses of treating children affected with **cerebral palsy.** H. 972, proposes an appropriation to the board of trustees of state institutions of higher learning for the purpose of providing necessary equipment, teaching staff and other personnel and for general expenses of a **school of nursing** at the University of Mississippi. H. 981, proposes an appropriation to the board of trustees of state institutions of higher learning for the purpose of paying for scholarships for **advanced studies in nursing.**

Bill Enacted.—H 531, was approved March 27, 1952. It provides an appropriation to the state medical education board for the purpose of carrying out the provisions of the law creating the board and for the purpose of providing **scholarships for** bona fide citizens and residents of the state to secure for them **medical education** which would qualify them to become licensed and practicing physicians and surgeons in the state.

New Jersey

Bills Introduced.—A. 611, proposes to amend the licensing law applicable to private nursing homes and private hospitals so as to cover **convalescent homes** as well. S. 139, proposes the creation of a **board of chiropractic examiners** and defines chiropractice as a system of adjusting the articulations of the spinal column, by hand only, for the correction of a cause of disease. Licentiates under this act would have a right, in the examination of patients, to use all scientific instruments and x-ray necessary for the purpose of analysis, providing however that a license to practice chiropractic would not permit the holder to prescribe, administer, or dispense drugs in the treatment of any human ailment, disease, pain, injury, deformity, mental or physical condition, or to perform surgical operations or to perform obstetrics or to reduce or set fractures or dislocations.

Bill Enacted.—A. 1, has become Ch. 3 of the Laws of 1952. It provides an appropriation for the **construction of,** among other things, state mental and charitable **hospitals.**

New York

Bills Enacted.—S. 3, has become Ch. 354 of the Laws of 1952. It directs the mental health commission to formulate a program for the development and maintenance of one or more clinics operated within the state operated by public or private nonprofit community agencies for the diagnosis, treatment, and rehabilitation of **chronic alcoholics.** S. 2216, has become Ch. 413 of the Laws of 1952. It requires all schools under state control or supported wholly or in part by public money to teach the **nature of alcoholic drinks** and their effects on the human system in connection with the various divisions of physiology and hygiene.

Rhode Island

Bill Introduced.—H. 785, to amend the employment security act, proposes the appointment of a **state advisory council** to aid the administrator of employment security in formulating policies and solving problems relating to the administration of the employment security act and assuring him impartiality, neutrality, and freedom from partisan influence in the solution of such problems. One member of such council shall be a representative of the medical profession.

South Carolina

Bill Enacted.—H. 1775, has become Ratification No. 905 of the Acts of 1952. It provides regulations for the detention, guardianship, and treatment of mentally ill and **mentally deficient persons.**

Virginia

Bills Enacted.—H. 132, has become Ch. 451 of the Laws of 1952. It amends the uniform narcotic act by **defining "opium"** as including all synthetic drugs including addiction forming or addiction sustaining properties similar to morphine and so proclaimed by the President of the United States under the provisions of the Federal Narcotic Law. It also provides special penalities where the offense charge consists of the sale of marijuana or narcotics to a minor. H. 148, has become Ch. 452 of the Laws of 1952. It requires every practicing medical doctor in the state to obtain a **revenue license,** except those who were regularly practicing medicine prior to 1909 and interns in a licensed hospital during the 12 months next following their graduation from medical school.

MEDICAL NEWS

CALIFORNIA

Popular Medical Lectures.—Stanford University School of Medicine, San Francisco, is giving a series of Popular Medical Lectures at Lane Hall on Wednesday evenings. Remaining lectures are as follows:

April 23, The Age of Medical Miracles, Anthony J. J. Rourke.
May 7, Natural Childbirth—What It Is and What It Isn't, Lyman M. Stowe.

Traveling State Mental Hygiene Clinic.—A traveling state mental hygiene clinic opened in Chico in January as one of three being established in Northern California by the State Department of Mental Hygiene. It is reported to be the first traveling clinic in the state and makes psychiatric service available north of Sacramento. Other clinics are to be located in Marysville and Redding, with central offices in Chico. The staff includes a psychiatric social worker, a clinical psychologist, and consulting psychiatrist, who will serve the three clinics. Dr. Joseph H. Harris, consulting psychiatrist, is director of the state mental hygiene clinic in Sacramento and acting director of the traveling clinic.

COLORADO

Clinic at Grand Junction.—The Western Colorado Spring Clinic was held at Grand Junction April 18-19 at the LaCourt Hotel. Guest speakers included Drs. Maxwell M. Wintrobe and Thomas F. Keyes, Salt Lake City; Drs. William H. Mast and Robert O. Turek, Cleveland; Dr. Lawrence K. Gundrum, Los Angeles, and Dr. John Z. Bowers, dean of the University of Utah School of Medicine, Salt Lake City.

Seminar on Poliomyelitis.—The University of Colorado School of Medicine offers a seminar in poliomyelitis, to be held May 1-3. In addition to individual presentations, there will be panel discussions on "Etiology and Management of Pain and Spasm," "Other Diseases Simulating Polio—e. g., Coxsackie Infections, Encephalitides, Neuromuscular Disorders," and "When an Epidemic Strikes Your Community—Pueblo in 1951." Demonstrations in patient care on May 3 will include nursing, the bulbar-respiratory case, and physical therapy. The registration fee of $5 and tuition fee of $15 will be omitted for residents and interns of Colorado General and other affiliated hospitals and members of the faculty of the University of Colorado School of Medicine. Applications, accompanied by registration fee, should be sent to the Director of Postgraduate Education, University of Colorado School of Medicine, 4200 E. Ninth Ave., Denver 7.

CONNECTICUT

Record for Maternal Safety.—The State Department of Health reports that there were 42,355 live births in 1951 in Connecticut and only six maternal deaths. The resulting rate of 0.1 per 1,000 live births is claimed to be the lowest recorded to date. Of the children born in 1951 in Connecticut, 98.3% were born in the 32 general hospitals that provide maternity care.

DISTRICT OF COLUMBIA

Psittacosis Reported.—Dr. J. C. Eckhardt of the district department of health has reported a suspect case of psittacosis in an adult male. The patient became ill on March 16 with headache, cough, and signs of bronchopneumonia that responded to terramycin. There was a history of exposure to a sick parakeet. The bird, obtained in nearby Maryland on Feb. 14, died March 25. Psittacosis virus was isolated from tissues of the bird at the National Institutes of Health.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

FLORIDA

Annual Pediatric Seminar.—The annual seminar of the Dade County Pediatric Society will be held in Jackson Memorial Hospital, Miami, April 23-25. Dr. Aims C. McGuinness, dean, Graduate School of Medicine, University of Pennsylvania, will give a paper on "The Common Digestive Tract Disturbances of Infants and Children Including Steatorrhoeas: Diagnosis and Management." Dr. Jerome S. Harris, professor of pediatrics, Duke University, Durham, N. C., will discuss "ACTH and Cortisone—Latest Concepts with Review of the Indications, Contraindications and Dangers Inherent in Their Use." Registration fee for nonmembers is $10.

MASSACHUSETTS

Symposium on Cardiovascular Surgery.—The Alumni Association of the Boston University School of Medicine will hold its annual meeting May 3. Speakers, all of Boston, will include:

Joseph P. Lynch, Boston University, Management of Cardiac Trauma.
John W. Strieder, Boston University, Constrictive Pericarditis.
Dwight E. Harken, Harvard Medical School, Surgery of the Mitral Valve.
Robert E. Gross, Harvard Medical School, Treatment of Coarctation of the Aorta.

The speaker at the banquet will be Mr. Erwin D. Canham, whose subject will be "The Chances for Peace."

MISSOURI

Meeting in Columbia.—The consulting staff of the Ellis Fischel State Cancer Hospital in Columbia will meet on April 27 at 11 a. m. The scientific program is as follows:

Lauren V. Ackerman, St. Louis, End Results of Treatment in Cancer of the Breast.
Richard E. Johnson, Columbia, Design of a Clinical Experiment.
Henry Schwarz II, Columbia, Factors Influencing Results of Surgical Treatment of Peptic Ulcer.

The meeting is open to all members of the medical profession.

Meeting of Psychiatric Association.—The Mid-Continent Psychiatric Association will meet at the Hotel President, Kansas City, April 26-27. The president, Dr. Andrew B. Jones, St. Louis, will speak on "The Degenerative Diseases, with Emphasis on Amyotrophic Lateral Sclerosis." Other speakers include the following:

Charles E. Thompson, Ph.D., and W. Payton Kolb, North Little Rock, Ark., Group Psychotherapy in Association with Alcoholics Anonymous Program.
Robert F. Freeman, Topeka, Kan., Problem of Senile Custodial Patients—Solution for Kansas.
Leo H. Bartemeier, Detroit, Psychotherapy of Psychosomatic Disorders.
Daniel Blain, Washington, D. C., Use of the Handicapped in Defense Mobilization.
Charles F. Obermann, Oklahoma City, Control of Tuberculosis in State Hospitals of Oklahoma.
G. Wilse Robinson, G. Wilse Robinson Jr., Paul Hines, Louise B. Loewy, and Jack D. DeMott, Kansas City, Toxic Delirious Psychosis and the Mold Antibiotics.
William G. Reese, Little Rock, Ark., Clinical Application of Conditional Reflex Methodology.
Dexter M. Bullard, Rockville, Md., Clinical Problems in Administrative Psychiatry.
Francis J. Braceland, Hartford, Conn., Psychosomatic Medicine in English Literature.
John A. Grimshaw, Topeka, Kan., Occurrence of Methemoglobinemia During Insulin Coma.

MONTANA

Postgraduate Course.—The Maternal and Child Welfare Committee of the Montana Medical Association in cooperation with the state board of health will present a postgraduate course during the week of April 21 on the care of premature infants. Guest speakers will be Dr. John A. Lichty, associate professor of pediatrics, and Dr. Paul Bruns, associate professor of obstetrics, both of the University of Colorado School of Medicine, Denver, and Miss Greene, nursing supervisor of the premature unit at Colorado General Hospital. Classes will be conducted in Bozeman, April 21, Lewistown, April 22, Great Falls, April 23, Havre, April 24, Glasgow, April 25, and Miles City, April 26.

NEW JERSEY

Doctors' Chorus.—The third annual benefit concert by the Doctors' Chorus of the Essex County Medical Society in behalf of the Essex County Service for the Chronically Ill will be given in the auditorium of the Mutual Benefit Life Insurance Company, Newark, on April 30 at 8:30 p. m.

NEW YORK

Postgraduate Courses.—Postgraduate instruction, provided at the request of the Saratoga County Chapter, Academy of General Practice, in conjunction with the Saratoga County Medical Society, is being given by the Medical Society of the State of New York in cooperation with the New York State Department of Health on Thursdays at the Hotel Rip Van Dam, Saratoga Springs. The remaining presentations are as follows:

April 24, 8:30 p. m., Leo Taran, Roslyn, L. I., Management of Rheumatic Fever.
May 1, 4:30 p. m., Frederick W. Williams, New York, Gangrene and the Management of the Surgical Diabetic.
May 22, 4:30 p. m., Irwin Wellen, New York, Hypertension, Edema, and Proteinuria in Pregnancy.

Woman's Auxiliaries Aid Nurses.—The Woman's Auxiliary to the Medical Society of the State of New York reports that during the past six years more than $32,000 has been raised to train 129 women in 24 counties in nursing education. This year five more counties will join the scholarship program started in 1946. Twenty-five nurses have graduated, and four students are scheduled to complete their courses this year as a result of the scholarships. Primary requirements for the scholarships are financial need, ability, and county residency. Several county auxiliaries have engaged in other activities beneficial to the nursing profession. One county established a $600 assistance fund to enable nurses to continue their education. Another awarded prizes to outstanding nursing students at hospital capping ceremonies, while others enlisted nursing recruits. Three other auxiliaries augmented their scholarship contributions with emergency funds.

New York City

Annual Kaplan Lecture.—The annual meeting of the Bellevue Hospital Radiation Therapy Alumni Association will be held May 8 at 1 p. m. in the eighth floor lecture hall of the administration building at Bellevue Hospital. The program will feature the annual Ira I. Kaplan Lecture, which will be given by Dr. James A. Corscaden, on "Treatment of Carcinoma of the Uterus." Members of the medical profession are invited.

Society News.—Dr. Henry L. Jaffe of the Hospital for Joint Diseases will present a paper on "Giant-Cell Tumor, Traumatic Bone Cyst, and Fibro-Osseous Lesions of Jaws" at the monthly conference of the New York Institute of Clinical Oral Pathology April 28 at the New York Academy of Medicine, room 440, at 9 p. m.

Montefiore Hospital Lectures.—The division of neoplastic diseases at Montefiore Hospital for Chronic Diseases is giving semimonthly lectures, with alternate meetings devoted to presentation of cases with discussion. On May 2, Dr. Joe V. Meigs, Boston, will speak on "Surgical Treatment of Cancer of the Cervix." On June 6, Dr. Arthur Purdy Stout will discuss "Tumors of Soft Tissue Origin."

Hospital Alumni Meeting.—The annual dinner and show of the Alumni Society of the Jewish Hospital of Brooklyn will be held May 4 beginning at 3 p. m. in Louria Auditorium. Subjects of papers to be presented are: "Demonstration—New Method for Determining Early Cardiac Enlargement," "Studies on Prematurity," "Histamine in Allergy," "Clinical Applications of the Ballistocardiograph," and "Practical Considerations in Electrolyte Therapy for the Surgical Patient."

Personals.—Dr. George T. Pack recently lectured before the faculty and medical student body of the University of Santo Domingo. He was awarded the Order of Merit of Juan Pablo Duarte by President Rafael Trujillo of the Dominican Republic, and was made an honorary member of the Dominican Medical Society.——Dr. Jacob J. Golub recently retired as medical director and executive vice-president of the Hospital for Joint Diseases after an affiliation of 23 years.

Courses in Resuscitation.—The Society for the Prevention of Asphyxial Death, Inc. is conducting a course in resuscitation in the Academy of Sciences Building, May 4-5 and June 6-7. The course is given regularly in New York the first Friday and Saturday of each month; out-of-town classes have met in Boston, Columbus, Washington, D. C., Los Angeles, and Honolulu. Requests for out-of-town courses will be considered if application is made by a group of at least 12 students. For information, address Secretary, Society for the Prevention of Asphyxial Death, Inc., 2 E. 63rd St., New York 21.

NORTH CAROLINA

Appointments to Wake Forest.—Three faculty members of the Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, have been promoted to full professors: Dr. Fred K. Garvey, urology; Dr. James A. Harrill, otolaryngology, and Dr. Robert L. McMillan, clinical internal medicine. Dr. Paul Haun, presently chief of the hospital construction unit of the psychiatry and neurology division of the Veterans Administration, has been appointed assistant professor of psychiatry and clinical director of Graylyn, Bowman Gray School of Medicine, a nervous and mental hospital.

OHIO

New Mental Hygiene Chief.—Dr. Calvin L. Baker, commissioner of mental hygiene since 1949, has resigned to resume private practice in Columbus and Dr. Lowell O. Dillon, assistant commissioner of mental hygiene, will succeed him.

Appoint Professor of Pediatrics.—Dr. William M. Wallace, assistant professor of pediatrics at Harvard Medical School, Boston, has been named director of Babies and Childrens Hospital, Cleveland, to succeed Dr. Charles F. McKahnn, now medical director of United Cerebral Palsy Associations. Dr. Wallace will also become professor of pediatrics at Western Reserve University School of Medicine.

Symposium on Cortisone and Compound F.—A symposium on cortisone and compound F, their local tissue actions, and possible mechanisms will be presented May 1 by the University of Cincinnati College of Medicine. There is no fee for the conference. Out-of-state speakers include:

Max Tishler, Ph.D., Rahway, N. J., Gregory G. Pincus, Sc.D., Shrewsbury, Mass., Chemistry of Cortisone and Compound F.
Ellis H. Steffensen, Detroit, Clinical Investigative Studies of the Local Use of Cortisone and Compound F in Diseases of the Eye.
Gregory G. Pincus, Shrewsbury, Mass., Joseph Lee Hollander, Philadelphia, Local Use of Cortisone and Compound F in Joints.
Charles West, New York, Studies of the Comparative Metabolic Effects of Cortisone and Compound F.
H. Dalton Jenkins, Boston, Comparative Clinical Studies of Cortisone and Compound F.
Augustus Gibson, Rahway, N. J., Summary of Preliminary Findings with Compound F and Need for Future Studies.

PENNSYLVANIA

Tuberculosis Society Meeting.—The annual meeting of the Pennsylvania Tuberculosis and Health Society will be held April 28-30 at the Bellevue-Stratford Hotel, Philadelphia. The program will emphasize the problem of controlling tuberculosis and improving public health services in Pennsylvania. The Pennsylvania Trudeau Society will meet at the same time, its scientific sessions being held Tuesday, April 29. "What the Future Holds for Public Health in Pennsylvania" will be discussed by Dr. Russell E. Teague, Philadelphia, state secretary of health, at the forenoon session, April 30.

Philadelphia

Open House at Health Center.—At its open house April 23 the Fife-Hamill Memorial Health Center will present exhibits and demonstrations on its many activities, including child health, dental hygiene, public health nursing, venereal disease control, health education, nutrition service, and mass x-ray screening. Dr. Bernard M. Blum, professor of public health at Jefferson Medical College of Philadelphia, is director of the center. A health maintenance clinic is headed by Dr. Gulden Mackmull, assistant professor of preventive medicine at Jefferson.

Philadelphia Fellowship Award.—Dr. Pascal F. Lucchesi, who recently resigned as superintendent and medical director of the Philadelphia General Hospital to become executive vice-president of the new Einstein Medical Center, received the 1952 Philadelphia Fellowship Award on Feb. 6 at the annual dinner of the commissioners for "his efforts to achieve democratic training, personnel and admission practices at Philadelphia General Hospital." The award is made annually for outstanding contributions to better understanding and equality of treatment and opportunity for all racial, religious, and national groups.

Cancer Day Program.—The Academy of General Practice and the Cancer Control Committee of the Philadelphia County Medical Society will hold a joint Cancer Day program on April 23. Moving pictures from 3 to 4 p. m. will be followed by a panel on diagnostic methods useful in cancer. The participants are as follows:

Walter H. Maloney, Laryngeal Mirror.
Theodore P. Eberhard, Office Biopsy.
Mary D. Pettit, Vaginal Speculum.
Ralph Jones Jr., Laboratory Tests.
Harry E. Bacon, Sigmoidoscope.

Dr. J. Herbert Nagler, president of the Philadelphia Academy of General Practice, will serve as moderator. The discussion will be followed by a dinner at 6 p. m.

RHODE ISLAND

Isaac Ray Medical Library.—On March 5 this library was dedicated at Butler Hospital in Providence. The project, conceived by Dr. Gregory Zilboorg of New York, consultant in research and psychotherapy at Butler Hospital, received generous support from the Aquinas Fund. The new quarters were designed and built entirely by hospital personnel. Books on medical psychology from the libraries of the late Drs. Isaac Ray and G. Alder Blumer and other former superintendents will, it is anticipated, be part of a 10,000 volume library. So that the library will be equipped to meet more than local needs, a catalog of holdings of other psychiatric libraries in this country will be compiled.

SOUTH CAROLINA

College Resumes Original Name.—The Medical College of the State of South Carolina, Charleston, after 120 years has resumed by legislative action its original name, the Medical College of South Carolina.

TEXAS

Grant for Research.—The U. S. Public Health Service has awarded a grant of $13,296 to Dr. Andres Goth, professor of pharmacology, at Southwestern Medical School of the University of Texas, Houston, for a two year tissue metabolic study in experimental hypersensitivity.

State Medical Meeting in Dallas.—The annual session of the Texas Medical Association will be held in Dallas May 4-7, with headquarters at the Adolphus Hotel under the presidency of Dr. Allen T. Stewart of Lubbock. Guest speaker at the opening general meeting will be Dr. Robert P. Glover, Philadelphia, whose subject will be "Advances in Treatment of Stenotic Valvular Disease of the Heart." At the general meeting luncheon on Wednesday Mr. Arthur L. Conrad, Chicago, will speak on "The Key to Peace." Related organizations will meet Monday morning. There will be three clinical luncheons to accommodate nine specialty groups, and these will be followed by section meetings. Both alumni and fraternity banquets have been scheduled, and scientific and technical exhibits will be shown.

Preprofessional Health Education Conference.—A southwest regional Conference on Pre-Professional Health Education will be held May 15-17 at the University of Texas Medical Branch, Galveston, under the auspices of the Texas Academy of Medicine, the National Honorary Pre-Medical Society, and the Medical Branch. Professional schools, colleges, and universities in Arkansas, Louisiana, Oklahoma, and Texas will discuss opportunities for training in medicine, dentistry, pharmacy, veterinary medicine, public health, nursing, medical and radiation technology, physical and occupational therapy, hospital administration, medical case work, clinical psychology, and medical record librarianship. Representatives from national organizations in the health fields will participate.

VIRGINIA

War Memorial Fund Students.—The Richmond County Medical Society announces that three children of physicians who gave their lives in World War II were enrolled in college this fall under its War Memorial Fund. The latest additions to the nine students previously enrolled are Franklin D. R. Wald, son of Dr. Oscar Wald, Kings County; Judith Silberstein, daughter of Dr. Harry A. Silberstein, Rockland County, and Edith Evelyn Lyons, daughter of Dr. Alfred L. Lyons, Queens County.

WASHINGTON

Society Meeting.—The annual meeting of the Spokane Surgical Society will be held at the Davenport Hotel April 26. The guest speaker will be Dr. John H. Gibbon Jr., professor of surgery and director of surgical research, Jefferson Medical College, Philadelphia.

WEST VIRGINIA

Close VD Clinic in South Charleston.—After treating over 20,000 persons for venereal diseases since 1944, the West Virginia Medical Center in South Charleston was closed permanently on April 15, the last patient having been admitted March 31. State health officials feel that because of the great decrease in venereal diseases, the cost of operating the center does not justify its continuance. When the clinic was opened in 1944, the average daily patient load was between 150 and 200 persons as compared to the present patient load of from 20 to 30 persons. The work of the center will be taken over by 10 treatment centers over the state. Eight of the clinics will be located in southern West Virginia and two in the northern part of the state.

Public Health Association Meeting.—The 28th conference of the West Virginia Public Health Association will be held at the Daniel Boone Hotel in Charleston May 8-9 under the presidency of Dr. David C. Prickett, Weston. Out-of-state speakers include:

Carl E. Buck, Dr.PH, Ann Arbor, Mich., Needed Public Health Legislation in West Virginia.
Seward E. Miller, Washington, D. C., Public Health Laboratory Services in the United States.
Vergil N. Slee, Hastings, Mich., The People, The Doctor, and the Health Department.
Miss Elizabeth Lovell, Raleigh, N. C., Mental Health as Related to Health Education in the Community.
Mrs. Phillip E. Nelbach, New York, Community Health Councils.
Miss Pearl McIver, Washington, D. C., Nursing in an Expanding Public Health Program.

WISCONSIN

Lovenhart Lecture.—Under the auspices of Phi Delta Epsilon, Dr. Roy R. Grinker, director of the Institute for Psychosomatic and Psychiatric Research and Training, Michael Reese Hospital, Chicago, will speak at the University of Wisconsin in the auditorium of the Service Memorial Institute at 8 p. m. April 29 on "Irritability, Tension, and Anxiety."

ALASKA

Dr. Blomquist Heads Research Center.—Dr. Edward T. Blomquist has arrived in Anchorage to take up his duties as medical officer in charge of the Arctic Health Research Center of the U. S. Public Health Service. He succeeds Dr. Jack C. Haldeman, who was transferred to Washington as chief of the Division of State Grants, Bureau of States Services, U. S. Public Health Service.

Health Ships Begin Programs.—The *Motor Vessel Health* left Seward Feb. 22 to offer its generalized health program in isolated villages along the Aleutian chain and the Bering Sea. The health ship expects to be in St. Lawrence Island ports by June, and then cross to Norton Sound, working its way back southward with

calls at the coastline villages. Dr. Hazel I. Blair is the *Health's* physician in charge for the fourth year, and Miss Jeanne Schaver is again aboard as public health nurse.

The *Motor Ship Hygiene,* after a trip to dry dock for repairs, resumed operation in southeastern waters in February, with Klawock, Craig, Haines, and Hoonah first on its itinerary. It was scheduled to stop briefly in Juneau in mid-March. Dr. Lawrence R. Rose is physician in charge of the ship's generalized health program. Dr. Guy Yates is dentist, Mrs. Yates is dental assistant, and Irene Barnett is public health nurse.

GENERAL

Society Election.—At the annual meeting of the Central Surgical Association in Toronto, March 6-8, Dr. Richard K. Gilchrist of Chicago was elected president; Dr. James T. Priestley of Rochester, Minn., president-elect; Dr. Robert M. Zollinger of Columbus, Ohio, secretary, and Dr. Leon J. Leahy of Buffalo, treasurer.

Dr. Ralph S. Lillie Dies.—Ralph S. Lillie, Ph.D., professor emeritus of pharmacy and zoology at the University of Chicago, died in Albert Merritt Billings Hospital March 19, aged 76. Before joining the faculty in 1924, he had served at Nebraska, Harvard, John Hopkins, and Pennsylvania universities. Dr. Lillie was internationally known for his work on the fundamental properties of living substances and the physiology of stimulation, growth, cell division, and radiation effects. He was an investigator and trustee of the Marine Biological Laboratory at Woods Hole, Mass.

Award for Research in Orthopedic Surgery.—A prize of $1,000, donated by the Kappa Delta Sorority, will be awarded annually by the American Academy of Orthopaedic Surgeons for the best research related to orthopedic surgery and performed by an American citizen in the United States. This research must be presented to the committee on scientific investigation of the academy before Nov. 1. Researchers interested in competing for this prize are requested to secure information from Dr. Claude N. Lambert, 104 S. Michigan Ave., Chicago 3, Chairman of the Award Committee for 1952.

Seminar on Venereal Disease.—Physicians, nurses, social workers, and other interested persons are invited to attend a seminar on venereal diseases, May 21-22 at the Hotel Bradford, Boston. The seminar, which is sponsored by the Public Health Service in conjunction with state health departments of the New England and Middle Atlantic States, will devote one day to clinical discussion and one to public health techniques of controlling venereal disease. Dr. Nicholas J. Fiumara, director of the division of venereal disease, Massachusetts Department of Public Health, is helping to make local arrangements.

Lectures on Athletics by Dr. Jokl.—Dr. Ernst Jokl, formerly chief research officer in physical education in the Union Education Department, Fredonia, South Africa, and now physician in chief, Sporthochschule, Cologne, is in the United States lecturing before medical schools and schools of physical education. Among other institutions, Dr. Jokl has delivered addresses at Columbia University, New York University, University of Illinois, University of Chicago, University of Wisconsin, and the University of Southern California. On April 5 he addressed the annual sessions of the American Academy of Physical Education in Los Angeles. Dr. Jokl's presentation covers the subject of the clinical physiology of athletics, a paper for which he has been awarded the Buckston-Browne Empire Prize of the Harvein Society of London.

Fellowships in Child Psychiatry.—Fellowships offering training in child psychiatry are available in a number of member clinics of the American Association of Psychiatric Clinics for Children. The training begins at a third year, postgraduate level with minimum prerequisites of graduation from medical school, a general or rotating internship, and a two year residency in psychiatry, all approved. This training is in preparation especially for positions in community clinics devoted wholly or in part to treatment of children with psychiatric problems on an outpatient basis. Most of the clinics have a two year training period, although a few will consider giving one year training in special cases. Fellowship stipends are about $3,600 and come mainly from the U. S. Public Health Service. Special arrangements may be made to supplement the stipends by taking on other responsibilities locally. A limited number of training centers can offer higher stipends. For application forms address Miss Mary C. Bentley, Executive Assistant, American Association of Psychiatric Clinics for Children, 1790 Broadway, Room 916, New York 19.

Muscular Dystrophy Association Meeting.—On May 17-18 Muscular Dystrophy Associations of America, Inc., will hold its annual medical conference at the Park Sheraton Hotel, New York City. Among those who will participate are Dr. Pearce Bailey, head of the National Institute of Neurological Diseases and Blindness; Dr. Albert Szent-Gyorgyi, director, Institute for Muscle Research; Roger J. Williams, Ph.D., University of Texas; Dr. Ade T. Milhorat, New York Hospital-Cornell Medical Center; Dr. Sidney Farber, Children's Hospital, Boston; Dr. H. Houston Merritt, Neurological Institute of Presbyterian Hospital; Dr. Joseph L. Lilienthal Jr., Johns Hopkins Hospital; Dr. Leona Baumgartner, New York City Department of Health; Karl E. Mason, Ph.D., University of Rochester; Dr. Augustus S. Rose, University of California; Dr. Thaddeus S. Danowski, University of Pittsburgh School of Medicine; Dr. S. M. Small, University of Buffalo; Dr. Arthur S. Abramson, Veterans Administration Hospital, New York; Wilfried F. H. M. Mommaerts, Ph.D., Duke University; Harry M. Hines, Ph.D., State University of Iowa College of Medicine, and Edwin L. Hove, Ph.D., Alabama Polytechnic Institute. Requests for information should be addressed to the Chairman, Medical Conference, Muscular Dystrophy Associations of America, 21 E. 40th St., New York 16.

FOREIGN

Meeting of Nobel Laureates.—The second meeting of Nobel laureates (prize winners in chemistry and physics) will be held June 23-27 at Lindau-Bodensee in Bavaria, Germany. Among the proposed subjects to be discussed are further development of chemotherapy of tuberculosis, curative success of chemistry and its restrictions in medicine, agents of growth and their antagonists, radiochemistry and the decomposition of uranium, and application of radioactive indicators for investigation of any process in animal bodies.

International Congress on Dietetics.—This congress is to be held in Amsterdam, The Netherlands, July 7-11 in the Royal Tropical Institute. All persons interested in nutrition and dietetics may become members of the congress on payment of fl. 30 ($30) to the general secretary, Miss Diane J. Ten Haaf at the Educational Bureau of the Nutrition Council, 42 Koninginnegracht, The Hague, The Netherlands. Languages of the congress will be English and French. During the morning sessions members will deliver lectures, and all afternoon sessions will be devoted to the subject "The Dietetic Profession and Its Problems."

Meeting of International College of Surgeons.—The first meeting of the French chapter of the International College of Surgeons will be held in Bordeaux, France, May 13-15. Prof. Dr. Raymond Darget, president of the French chapter, and Prof. Dr. Georges Portmann, dean of the Faculty of Medicine, Bordeaux, will officiate. Operative clinics will be held in Bordeaux hospitals and clinics, and papers will be presented on "Arterial Hypertension—Its Diagnosis and Treatment," "Surgical Injury of the Ureter," and "Laminectomy and Vertebral Grafts." Information concerning the meeting may be obtained from Prof. Dr. Raymond Darget, 17, rue Castéja, Bordeaux, France.

CORRECTION

Plastic Surgery of the Nose.—The retail price of Brown and McDowell's book on this subject was incorrectly stated in THE JOURNAL, March 1 and March 15, as being $10. The retail price of the book is $15.

Gastrointestinal Tract in the Aged.—In the article by this title in THE JOURNAL, March 29, 1952, in the 11th line on page 1079, the dosage in parentheses "(20 to 30 gm.)" should read "(200 to 300 mg.)."

MEETINGS

American Medical Association, Chicago, June 9-13. Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secretary.

American Academy of Pediatrics, Areal Meeting, Hotel Statler, Washington, D. C., May 22-24. Dr. E. H. Christopherson, 636 Church St., Evanston, Ill., Executive Secretary.

American Academy of Neurology, Brown Hotel, Louisville, Ky., Apr. 24-25. Dr. Francis M. Forster, 3800 Reservoir Rd. N.W., Washington, D. C., Secretary.

American Association for Thoracic Surgery, Dallas, Texas, May 8-10. Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif., Secretary.

American Association of the History of Medicine, Kansas City, Kans., May 1-3. Dr. Iago Galdston, 2 East 103d St., New York 29, Secretary.

American Association on Mental Deficiency, Philadelphia, May 20-24. Dr. Neil A. Dayton, P. O. Box 96, Willimantic, Conn., Secretary.

American Blood Irradiation Society, Conrad Hilton Hotel, Chicago, June 7-8. Dr. H. T. Lewis, 5230 Center Ave., Pittsburgh, Secretary.

American Broncho-Esophagological Association, Royal York Hotel, Toronto, Canada, May 20-21. Dr. Edwin N. Broyles, 1100 North Charles St., Baltimore 1, Secretary.

American College of Chest Physicians, Congress Hotel, Chicago, June 5-8. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago, Executive Secretary.

American College Health Association, Copley Plaza Hotel, Boston, May 1-3. Miss Edith M. Lindsay, School of Public Health, University of California, Berkeley 4, Calif., Secretary.

American College of Physicians, Cleveland, April 21-25. Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Executive Secretary.

American College of Surgeons, Sectional Meetings:
Toronto, Canada, Royal York Hotel, May 15-17. Dr. Stuart D. Gordon, 170 St. George St., Toronto 5, Canada, Chairman.

American Dermatological Association, Broadmoor Hotel, Colorado Springs, Colo., Apr. 23-26. Dr. Louis A. Brunsting, 102 Second Ave. S.W., Rochester, Minn., Secretary.

American Electroencephalographic Society, Hotel Claridge, Atlantic City, N. J., May 10-11. Dr. John A. Abbott, Massachusetts General Hospital, Boston 14, Secretary.

American Federation for Clinical Research, Haddon Hall, Atlantic City, N. J., May 4. Dr. Stewart Wolf, 525 East 68th St., New York 21, Secretary.

American Gastro-Enterological Association, Hotel Claridge, Atlantic City, N. J., May 2-3. Dr. Dwight L. Wilbur, 655 Sutter St., San Francisco 2, Secretary.

American Gastroscopic Society, Hotel Claridge, Atlantic City, N. J., May 2-3. Dr. H. Marvin Pollard, University Hospital, Ann Arbor, Mich., Secretary.

American Geriatrics Society, Hotel LaSalle, Chicago, June 5-7. Dr. Malford W. Thewlis, 25 Mechanic St., Wakefield, R. I., Secretary.

American Goiter Association, Chase Hotel, St. Louis, May 1-3. Dr. George C. Shivers, 100 East St. Vrain St., Colorado Springs, Colo., Secretary.

American Gynecological Society, The Homestead, Hot Springs, Va., May 12-14. Dr. John I. Brewer, 104 South Michigan Ave., Chicago, Secretary.

American Heart Association, Hotel Statler, Cleveland, April 17-20. Dr. Willian H. Bunn, 1775 Broadway, New York 19, Secretary.

American Laryngological Association, Royal York Hotel, Toronto, Canada, May 23-24. Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Secretary.

American Laryngological, Rhinological and Otological Society, Royal York Hotel, Toronto, Canada, May 20-22. Dr. C. Stewart Nash, 277 Alexander St., Rochester 7, N. Y., Secretary.

American Medical Women's Association, Chicago, June 8. Dr. Irma Henderson-Smathers, 610 Medical Bldg., Asheville, N. C., Corresponding Secretary.

American Neurological Association, Hotel Claridge, Atlantic City, N. J., May 8-10. Dr. H. Houston Merritt, 710 West 168th St., New York 32, Secretary.

American Ophthalmological Society, Hot Springs, Va., June 5-7. Dr. Maynard C. Wheeler, 30 West 59th St., New York 19, Secretary.

American Otological Society, Royal York Hotel, Toronto, Canada, May 18-19. Dr. John R. Lindsay, 950 East 59th St., Chicago 37, Secretary.

American Pediatric Society, Hotel Chamberlin, Old Point Comfort, Va., May 7-9. Dr. Aims C. McGuiness, 237 Medical Laboratories, Univ. of Pennsylvania, Philadelphia 4, Secretary.

American Proctologic Society, Milwaukee, June 4-7. Dr. Stuart T. Ross, 131 Fulton Ave., Hempstead, L. I., N. Y., Secretary.

American Psychiatric Association, Atlantic City, N. J., May 12-16. Dr. R. Finley Gayle Jr., 6300 Three Chopt Rd., Richmond 21, Va., Secretary.

American Psychoanalytic Association, Chalfonte-Haddon Hall, Atlantic City, N. J., May 8-11. Dr. LeRoy M. A. Maeder, 1910 Rittenhouse Square, Philadelphia 3, Secretary.

American Radium Society, Chicago, June 8. Dr. John Wirth, 635 Herkimer St., Pasadena 1, Calif., Secretary.

American Rheumatism Association, Conrad Hilton Hotel, Chicago, June 6-7. Dr. William H. Kammerer, 33 East 61st St., New York 21, Secretary.

American Society for Clinical Investigation, Atlantic City, N. J., May 5. Dr. William M. M. Kirby, Univ. of Washington School of Medicine, Seattle 5, Secretary.

American Therapeutic Society, Blackstone Hotel, Chicago, June 5-8. Dr. Oscar B. Hunter Jr., 915 Nineteenth St. N.W., Washington 6, D. C., Secretary.

American Trudeau Society, Hotel Statler, Boston, May 26. Dr. John D. Steele, 1790 Broadway, New York 19, Secretary.

American Venereal Disease Association, Washington, D. C., May 1-2. Dr. William L. Fleming, 750 Harrison Ave., Boston 18, Secretary.

Arizona Medical Association, Hotel Westward Ho, Phoenix, April 30-May 3. Dr. Frank J. Milloy, 15 E. Monroe St., Phoenix, Secretary.

Arkansas Medical Society, Little Rock, April 21-23. Dr. William R. Brooksher, 215 Kelley Bldg., Fort Smith, Secretary.

Association of American Physicians, Atlantic City, N. J., May 6-7. Dr. Henry M. Thomas Jr., 1201 North Calvert St., Baltimore 2, Secretary.

California Medical Association, Biltmore Hotel, Los Angeles, April 27-30. Dr. Albert C. Daniels, 450 Sutter St., San Francisco 8, Secretary.

Catholic Hospital Association of the United States and Canada, Cleveland, May 26-29. Rev. John J. Flanagan, S.J., 1438 South Grand Blvd., St. Louis 4, Director.

Connecticut State Medical Society, Hartford, April 29-May 1. Dr. Creighton Barker, 160 St. Ronan Street, New Haven, Secretary.

Florida Medical Association, Hollywood, April 27-30. Dr. Samuel M. Day, P. O. Box 1018, Jacksonville, Secretary.

Georgia, Medical Association of, Atlanta-Biltmore Hotel, Atlanta, May 12-14. Dr. David H. Poer, 875 West Peachtree St. N.E., Atlanta, Secretary.

Hawaii Territorial Medical Association, Honolulu, May 1-4. Dr. I. L. Tilden, 510 S. Beretania St., Honolulu 13, Secretary.

Illinois State Medical Society, Chicago, May 13-15. Dr. Harold M. Camp, 224 South Main St., Monmouth, Secretary.

Industrial Medical Association, Cincinnati, April 21-24. Dr. Edward C. Holmblad, 28 E. Jackson Blvd., Chicago 4, Managing Director.

Iowa State Medical Society, Des Moines, April 28-30. Dr. Allan B. Phillips, 406 Sixth Avenue, Des Moines 9, Secretary.

Kansas Medical Society, Town House, Kansas City, April 27-May 1. Dr. Dale D. Vermillion, 512 New England Bldg., Topeka, Secretary.

Louisiana State Medical Society, Shreveport, April 28-30. Dr. C. Grenes Cole, 1430 Tulane Ave., New Orleans 12, Secretary.

Maryland, Medical and Chirurgical Faculty of the State of, Baltimore, April 28-30. Dr. George H. Yeager, 1211 Cathedral St., Baltimore 1, Secretary.

Massachusetts Medical Society, Hotel Statler, Boston, May 20-22. Dr. H. Quimby Gallupe, 22 Fenway, Boston 15, Secretary.

Mid-Continent Psychiatric Association, President Hotel, Kansas City, Mo., April 26-27. Dr. Paul Hines, 2625 W. Paseo, Kansas City, Mo., Secretary.

Minnesota State Medical Association, Minneapolis, May 26-28. Dr. B. B. Souster, 496 Lowry Medical Arts Bldg., St. Paul 2, Secretary.

Mississippi State Medical Association, Jackson, May 13-15. Mr. Roland B. Kennedy, 508 First Federal Bldg., Jackson, Executive Secretary.

National Tuberculosis Association, Copley Plaza and Statler Hotels, Boston, May 26-30. Mr. Kemp D. Battle, 1790 Broadway, New York 19, Secretary.

Nebraska State Medical Association, Cornhusker Hotel, Lincoln, May 12-15. Dr. R. B. Adams, 1315 Sharp Bldg., Lincoln 8, Secretary.

New Jersey, Medical Society of, Haddon Hall, Atlantic City, May 19-21. Dr. Marcus H. Greifinger, 315 West State St., Trenton 8, Secretary.

New Mexico Medical Society, Carlsbad, May 8-10. Dr. L. G. Rice Jr., 221 West Central Ave., Albuquerque, Secretary.

New York, Medical Society of the State of, Hotel Statler, New York, May 12-16. Dr. Walter P. Anderton, 292 Madison Ave., New York 17, Secretary.

North Carolina, Medical Society of the State of, Hotel Carolina, Pinehurst, May 5-7. Dr. Millard D. Hill, 203 Capitol Club Bldg., Raleigh, Secretary.

Ogden Surgical Society, Orpheum Theater, Ogden, Utah, May 21-23. Dr. George M. Fister, Ogden, Utah, President.

Ohio State Medical Association, Cleveland, May 20-22. Mr. Charles S. Nelson, 79 East State St., Columbus 15, Executive Secretary.

Oklahoma State Medical Association, Oklahoma City, May 19-21. Mr. R. H. Graham, 1227 Classen St., Oklahoma City, Executive Secretary.

Rhode Island Medical Society, Providence, May 6-8. Dr. Morgan Cutts, 106 Francis St., Providence 3, Secretary.

Society for Investigative Dermatology, Hotel Sherman, Chicago, June 7-8. Dr. Herman Beerman, 255 South 17th St., Philadelphia 3, Secretary.

Society for Pediatric Research, Old Point Comfort, Va., May 5-6. Dr. Sydney S. Gellis, 300 Longwood Ave., Boston 15, Secretary.

Society of American Bacteriologists, Hotel Statler, Boston, April 27-May 1. Dr. Henry W. Scherp, 260 Crittenden Blvd., Rochester 20, N. Y., Secretary.

SOCIETY OF BIOLOGICAL PSYCHIATRY, Hotel Claridge, Atlantic City, N. J., May 11. Dr. George N. Thompson, 1136 West Sixth St., Los Angeles 17, Secretary.

SOUTH CAROLINA MEDICAL ASSOCIATION, Ocean Forest Hotel, Myrtle Beach, May 13-15. Dr. N. B. Heyward, 120 West Cheves St., Florence, Secretary.

SOUTH DAKOTA STATE MEDICAL ASSOCIATION, Sioux Falls, May 18-20. Dr. L. J. Pankow, 302 First National Bank Bldg., Sioux Falls, Secretary.

SOUTHERN OREGON MEDICAL SOCIETY, Roseburg, May 13. Dr. F. C. Adams, 1435 Esplanade St., Klamath Falls, Secretary.

TEXAS MEDICAL ASSOCIATION, Dallas, May 4-7. Dr. Sam N. Key Sr., 700 Guadalupe St., Austin 3, Secretary.

THE ENDOCRINE SOCIETY, Palmer House, Chicago, June 5-7. Dr. Henry H. Turner, 1200 North Walker St., Oklahoma City 3, Secretary.

WESTERN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS, Biltmore Hotel, Los Angeles, April 26. Dr. David D. Holaday, American Can Company, Third and 20th Sts., San Francisco 7, Secretary.

WYOMING STATE MEDICAL SOCIETY, Nobel Hotel, Lander, June 5-7. Dr. Glenn W. Koford, 2020 Carey Ave., Cheyenne, Secretary.

INTERNATIONAL

AUSTRALASIAN MEDICAL CONGRESS, Melbourne Victoria, Australia, Aug. 22-30. Dr. C. H. Dickson, Medical Society Hall, 426 Albert St., East Melbourne, Victoria, Australia, General Secretary.

COMMONWEALTH AND EMPIRE HEALTH AND TUBERCULOSIS CONFERENCE, Central Hall, London, England, July 8-13. Dr. J. H. Harley Williams, Tavistock House North, Tavistock Sq., London, W.C.1, England, Secretary General.

CONGRESS OF THE INTERNATIONAL DIABETES FEDERATION, Leyden, Netherlands, July 7-12. Dr. F. Gerritzen, 33, Prinsegracht, The Hague, Netherlands, Secretary.

EUROPEAN CONGRESS OF CARDIOLOGY. University of London, Bloomsbury, W.C.1, England, Sept. 10-12. Dr. K. Shirley Smith, 35 Wimpole St., London, W.1, England, Secretary.

EUROPEAN SOCIETY OF CARDIOVASCULAR SURGERY, Strasbourg, France, Oct. 3-4. Sir James Learmonth, Department of Surgery, University New Buildings, Edinburgh 8, Scotland.

INTERAMERICAN CARDIOLOGICAL CONGRESS, Buenos Aires, Argentina, Aug. 31-Sept. 7. Dr. Blas Moia, Larrea 1132, Buenos Aires, Argentina, Secretary-General.

INTER-AMERICAN CONGRESS OF PUBLIC HEALTH, Havana, Cuba, Sept. 27-Oct. 1. Pan-American Sanitary Bureau, 2001 Connecticut Ave., Washington, D. C.

INTER-AMERICAN CONGRESS OF RADIOLOGY, Mexico City, Mexico, Nov. 2-8. Dr. Guido Torres Martinez, Marsella No. 11, Mexico, D.F., Secretary-General.

INTERNATIONAL COLLEGE OF SURGEONS, Madrid, Spain, May 20-24. Dr. Max Thorek, 850 West Irving Park Road, Chicago, Ill., U. S. A., Secretary-General.

INTERNATIONAL CONGRESS OF BIOCHEMISTRY, Paris, France, July 21-27. Prof. Jean Courtois, 4, Avenue de l'Observatoire, Paris 6e, France, Secretary.

INTERNATIONAL CONGRESS OF COMPARATIVE PATHOLOGY, Madrid, Spain, May 4-11. Prof. Carda-Aparici, Calle Fernando VI.8, Madrid, Spain, Secretary.

INTERNATIONAL CONGRESS OF DERMATOLOGY AND SYPHILOLOGY, London, England, July 21-26. Dr. Gordon B. Mitchell-Heggs, 5, Lisle St., London, W.C.2, England, General Secretary.

INTERNATIONAL CONGRESS ON DIABETES, Leyden, The Netherlands, July 7-12. Dr. F. Gerritzen, 33, Prinsegracht, The Hague, Netherlands.

INTERNATIONAL CONGRESS OF DIETETICS, Royal Tropical Institute, Amsterdam, The Netherlands, July 7-11. Miss Diane J. Ten Haaf, Educational Bureau of the Nutritional Council, 42 Koninginnegracht, The Hague, Netherlands, General Secretary.

INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST, Rio de Janeiro, Brazil, Aug. 24-30. Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Illinois, Executive Secretary.

INTERNATIONAL CONGRESS OF HAEMATOLOGY, Mar del Plata, Argentina, Sept. 21-26. Dr. Carlos Reussi, Anchorena 1710, Buenos Aires, Argentina, Secretary.

INTERNATIONAL CONGRESS OF HEALTH TECHNICIANS, Palais de la Mutualite, Paris, France, June 9-14. Secretariat General, 6 Square Desaix, Paris XVe, France.

INTERNATIONAL CONGRESS OF THE HISTORY OF MEDICINE, Nice-Cannes, France, and Monaco, Sept. 8-18. Dr. F. A. Sondervorst, 124 Avenue des Allies, Louvain, Belgium, Secretary-General.

INTERNATIONAL CONGRESS OF INTERNAL MEDICINE, Friends House, London, N.W.1, Eng. Sept. 15-18. Sir Harold Boldero, 12 Pall Mall East, London, S.W.1, England, Secretary.

INTERNATIONAL CONGRESS ON MEDICAL RECORDS, London, England, Sept. 7-12. Miss Gwen Perkins, 120 C Street S.E., Washington, D. C., Chairman.

INTERNATIONAL CONGRESS OF MEDICINE AND SPORT, Paris, France. May 29-June 1. Prof. Chailley-Bert, 1 rue Lacretelle, Paris 15e, France, Chairman.

INTERNATIONAL CONGRESS ON NEUROPATHOLOGY, Rome, Italy, Sept. 8-13. Dr. Armando Ferraro, 722 W. 168th St., New York, N. Y., U. S. A., Secretary General.

INTERNATIONAL CONGRESS OF OTO-NEURO-OPHTHALMOLOGY, Lisbon and Estoril, Portugal, April 22-26. Prof. Diogo Furtado, Servico de Neurologia, Hospital dos Capuchos, Lisbon, Portugal, General Secretary.

INTERNATIONAL CONGRESS OF PHYSICAL MEDICINE, London, England, July 14-19. Dr. A. C. Boyle, 45, Lincoln's Inn Fields, London, W.C.2, England, Hon. Secretary.

INTERNATIONAL CONGRESS OF PHYSIOPATHOLOGY OF ANIMAL REPRODUCTION AND ARTIFICIAL INSEMINATION, Copenhagen, Denmark, July 7-10. Prof. Ed. Sorensen, The Royal Veterinary and Agricultural College, Bülowsvej 13, Copenhagen V, Denmark.

INTERNATIONAL CONGRESS OF UROLOGY, The Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 15-18. Dr. John A. Taylor, 2 East 54th St., New York, N. Y., U. S. A., Secretary-General.

INTERNATIONAL SOCIETY OF GEOGRAPHIC PATHOLOGY, Liège, Belgium, July 15-18. Dr. Fred C. Roulet, Habelstr. 24, Basei, Switzerland, Secretary General.

INTERNATIONAL UNION AGAINST TUBERCULOSIS, Rio de Janeiro, Brazil, Aug. 24-27. Prof. Etienne Bernard, 66 Blvd. St. Michel, Paris 6e, France, Secretary-General.

NEURORADIOLOGIC SYMPOSIUM, Stockholm, Sweden, Sept. 17-20. Docent Ake Lindbom, Symposium Neuroradiologicum, Serafimerlasarettet, Stockholm K., Sweden, Secretary.

PAN-AMERICAN CONGRESS OF GASTROENTEROLOGY, Mexico City, D.F., May 11-17. Dr. R. Flores-Lopez, Calle Dr. Balmis, No. 148, Mexico City, D.F., Secretary.

PAN-EUROPEAN CONGRESS OF GASTROENTEROLOGY, Bologna, Florence and Rome, Italy, April 20-26. Prof. Guido Bassi, Policlinic of St. Orsola, Bologna, Italy, Secretary.

WORLD MEDICAL ASSOCIATION, Athens, Greece, Oct. 12-16. Dr. Louis H. Bauer, 2 East 103d St., New York 29, N. Y., Secretary General

EXAMINATIONS AND LICENSURE

NATIONAL BOARD OF MEDICAL EXAMINERS

NATIONAL BOARD OF MEDICAL EXAMINERS: *Part II*, April 21-22; *Parts I and II*, June 18-20; *Part I*, September. All centers where there are five or more candidates. Exec. Sec., Dr. John P. Hubbard, 225 S. 15th St., Philadelphia 2.

EXAMINING BOARDS IN SPECIALTIES

AMERICAN BOARD OF ANESTHESIOLOGY: Various Locations, July 18. Final date for filing application was Jan. 18. Sec., Dr. C. B. Hickcox, 80 Seymour St., Hartford 15.

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOLOGY: *Oral.* Chicago, May 1952. Final date for filing application was Feb. 1, 1952. Candidates must have completed three years of approved training prior to July 1, 1952. Sec., Dr. George M. Lewis, 66 E. 66th St., New York 21.

AMERICAN BOARD OF INTERNAL MEDICINE: *Written.* Various centers and overseas, Oct. 20. Closing date for the acceptance of applications for the written examination will be May 1. *Oral.* Chicago, June 5-7, San Francisco, September or October; New York City, November or December. Subspecialty: Gastroenterology. *Examination.* Chicago, June 7. Exec. Sec.-Treas., Dr. William A. Werrell, 1 West Main Street, Madison 3.

AMERICAN BOARD OF NEUROLOGICAL SURGERY: *Oral.* Chicago, May 1952. Final date for filing applications for the May 1952 examination was Jan. 1. Sec., Dr. W. J. German, 789 Howard Ave., New Haven 4, Conn.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY: *Oral.* Chicago, June 9-13, 1952. Final date for filing application was Feb. 1, 1952. Sec., Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland 6.

AMERICAN BOARD OF OPHTHALMOLOGY: *Practical.* Philadelphia, May 29-June 3; Chicago, Oct. 6-10 and New York City, June 6-10, 1953. Sec., Dr. Edwin B. Dunphy, 56 Ivie Road, Cape Cottage, Maine.

AMERICAN BOARD OF ORTHOPAEDIC SURGERY: *Part I.* San Francisco and Washington, D. C., April 25-26. Sec., Dr. Harold A. Sofield, 122 South Michigan Ave., Chicago 3.

AMERICAN BOARD OF OTOLARYNGOLOGY. Toronto, May 13-17; Chicago, Oct. 6-10. Sec., Dr. Dean M. Lierle, University Hospitals, Iowa City.

AMERICAN BOARD OF PEDIATRICS: *Oral.* Washington, D. C., May 2-4; San Francisco, June 27-29; Chicago, October and Boston, late November. Exec. Sec., Dr. John McK. Mitchell, 6 Cushman Road, Rosemont, Pa.

AMERICAN BOARD OF PHYSICAL MEDICINE AND REHABILITATION: *Parts I and II.* Chicago. June 8 and 9. Sec., Dr. Robert L. Bennett, Georgia Warm Springs Foundation, Warm Springs, Ga.

AMERICAN BOARD OF PLASTIC SURGERY: *Oral and Written.* St. Louis, May 17-19. Sec., Dr. Bradford Cannon, 4647 Pershing Ave., St. Louis 8.

AMERICAN BOARD OF PROCTOLOGY. *Part I.* Kansas City, Minneapolis, Philadelphia and San Francisco. May 3. Examination includes both oral and written examinations in the basic sciences. *Dates and places tentative.* Sec., Dr. Louis A. Buie, 102-110 2d Ave., S.W., Rochester, Minn.

AMERICAN BOARD OF PSYCHIATRY AND NEUROLOGY: Chicago, June 13-14. Final date for filing applications was March 15. New York City, Dec. 15-16. Final date for filing applications is Sept. 15. Sec.-Treas., Dr. David A. Boyd, Jr., 102-110 Second Ave. S.W., Rochester, Minn.

AMERICAN BOARD OF RADIOLOGY: *Oral.* Spring 1952. Final date for filing application was Dec. 31. Sec., Dr. B. R. Kirklin, 102-110 Second Ave., S.W., Rochester, Minn.

AMERICAN BOARD OF SURGERY: *Written.* Various Centers. October 1952. Sec., Dr. J. Stewart Rodman, 225 15th St., Philadelphia.

DEATHS

Kiger, William Henry ✠ Los Angeles; born in 1876; Toledo Medical College, 1900; professor emeritus of proctology at the College of Medical Evangelists; an Associate Fellow of the American Medical Association and in 1943 a member of its House of Delegates; in 1928 president, and for 12 years on the governing council of the California Medical Association; for 22 years on the governing council of the Los Angeles County Medical Association, and in 1924 president; specialist certified by the American Board of Surgery and the American Board of Proctology; member and past president of the American Proctologic Society; fellow of the American College of Surgeons; veteran of the Spanish-American War; member of the surgical staff, Los Angeles County Hospital; member of the staff, Hospital of the Good Samaritan, Methodist Hospital of Southern California, California and Cedars of Lebanon hospitals, and Presbyterian Hospital-Olmstead Memorial; died February 10, aged 75, of carcinoma of the prostate.

Levine, Eugene Boris ✠ Los Angeles; born in Lawrence, Mass., March 12, 1914; Louisiana State University School of Medicine, New Orleans, 1939; certified by the National Board of Medical Examiners; specialist certified by the American Board of Internal Medicine; member of the board of directors of the Los Angeles Heart Association; assistant professor of medicine at the College of Medical Evangelists; consultant in cardiology to the Los Angeles City Health Department; chief of the cardiac clinic of Cedars of Lebanon Hospital; senior attending physician at Los Angeles County Hospital; died Feb. 26, aged 37, of Hodgkin's disease.

Leeper, Edward Paul ✠ Dallas, Texas; University of Texas School of Medicine, Galveston, 1928; clinical assistant professor of medicine, Southwestern Medical School of the University of Texas; formerly on the faculty of Baylor University College of Medicine; fellow of the American College of Physicians; member of the American Heart Association and the American Diabetes Association; city health officer for University Park; medical director for Praetorian Life Insurance Company; served during World War II; affiliated with Parkland, Methodist and Medical Arts hospitals; died Feb. 9, aged 48, of a heart attack.

Oliphant, John William ✠ Medical Director, U. S. Public Health Service, Washington, D. C.; born in Fort Branch, Ind., July 6, 1901; Indiana University School of Medicine, Indianapolis, 1931; commissioned in the U. S. Public Health Service in 1932; formerly assistant director of the Rocky Mountain Laboratory of the U. S. Public Health Service in Hamilton, Mont.; from 1938 to 1946 assigned to the National Institute of Health in Bethesda, Md., where he was serving at the time of his death; member of the American Epidemiological Society; died Jan. 10, aged 50, of accidental carbon monoxide poisoning.

Ackermann, William ✠ Milwaukee; Wisconsin College of Physicians and Surgeons, Milwaukee, 1896; member of the National Gastroenterological Association; formerly on the faculty of Marquette University School of Medicine; died Jan. 26, aged 76, of arteriosclerosis, hypertension, and carcinoma of the stomach.

Alford, Redman Lee ✠ Vandalia, Mo.; Marion-Sims College of Medicine, St. Louis, 1896; served as mayor and member of the board of public works and school board; for several years member of the board of visitors of the University of Missouri, Columbia; affiliated with Audrain Hospital in Mexico; died Jan. 15, aged 79, of emphysema.

Allison, Jackson Way, York, Pa.; University of Pittsburgh School of Medicine, 1928; member of the American Medical Association; served during World War II; died Jan. 15, aged 50, of cerebral hemorrhage.

Bailey, Bayard Melvin, La Jolla, Calif.; University of Illinois College of Medicine, Chicago, 1923; died in Scripps Memorial Hospital Feb. 4, aged 55.

Buckner, Roy Alvan, Gilman, Ill.; Chicago College of Medicine and Surgery, 1911; member of the American Medical Association; served as mayor of Gilman; affiliated with Iroquois Hospital in Watseka; died Jan. 15, aged 68, of carcinoma of the intestine.

Clark, Hines ✠ Crowell, Texas; Fort Worth School of Medicine, Medical Department of Fort Worth University, 1900; health officer of Crowell and of Foard County; instrumental in establishing the Foard County Hospital, which was opened in 1941; his portrait was hung in the hospital lobby in December, 1951, on the 51st anniversary of his medical practice in the county; served during World War I; died in Wichita Falls Clinic Hospital, Wichita Falls, Jan. 22, aged 75, of coronary thrombosis.

Coleman, Joseph Gilbert, Hamburg, N. J.; Albany (N. Y.) Medical College, 1897; member of the American Medical Association; past president of the Sussex County Medical Society; president of the Hardyston National Bank; died in Franklin (N. J.) Hospital Feb. 2, aged 76, of obstructive jaundice due to inoperable carcinoma.

Coomes, M. Joseph, Shelbyville, Ind.; Louisville (Ky.) Medical College, 1892; member of the American Medical Association; served during World War I; died in the W. S. Major Hospital Jan. 6, aged 81, following a fracture of the femur received when struck by an automobile.

Davis, Egbert Frank, Wyandotte, Mich.; Detroit College of Medicine, 1908; member of the American Medical Association; on the staff of Wyandotte General Hospital; died in Harper Hospital, Detroit, Jan. 12, aged 75.

Dickie, Emmet H., Chatsworth, Ga.; Georgia College of Eclectic Medicine and Surgery, Atlanta, 1913; member of the American Medical Association; served as mayor and member of the county board of health; died Nov. 15, aged 65.

Dodd, William Lincoln, Amity, Pa.; Western Pennsylvania Medical College, Pittsburgh, 1888; died Jan. 18, aged 85, of senility.

Downey, Louis J., Vincennes, Ind.; Washington University School of Medicine, St. Louis, 1902; formerly physician for the Indiana Boys' School in Plainfield; died in Good Samaritan Hospital, Vincennes, Jan. 3, aged 76, of coronary infarction and arteriosclerosis.

Durley, Howard, Milan, Tenn.; Rush Medical College, Chicago, 1910; died in St. Mary's Hospital, Humboldt, Jan. 12, aged 75, of a cerebral accident, chronic myocarditis, and hypertension.

Eason, Samuel Echols, New Albany, Miss.; Memphis (Tenn.) Hospital Medical College, 1905; twice elected vice-president of the Mississippi State Medical Association, serving as councilor of the third district for four years; past president and secretary of the North Mississippi Medical Society; president of the state board of health and formerly county health officer; on the staff of New Albany Hospital, where he died Jan. 26, aged 72, of chronic myocarditis.

Evans, Arthur John ✠ Magdalena, N. M.; Louisville (Ky.) Medical College, 1905; formerly state senator and district health officer; a contract physician in the U. S. Army from 1937 to 1941; died in Albuquerque Jan. 28, aged 68, of carcinoma of the nasopharynx.

Finch, Risdon W., Defiance, Ohio; Starling Medical College, Columbus, 1880; past president and secretary-treasurer of the Defiance County Medical Society; died Jan. 25, aged 95, of senility.

Flax, Ira Isador, Newark, N. J.; University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, 1924; served during World War II; on the consulting staff of Veterans Administration Hospital in Lyons, on the staffs of Newark City and Beth Israel hospitals; died Jan. 26, aged 49, of coronary disease.

✠ Indicates Fellow of the American Medical Association.

Flick, Earl Joseph, Royal Oak, Mich.; Wayne University College of Medicine, Detroit, 1937; member of the American Medical Association; served during World War II; died Jan. 24, aged 40.

Frey, Emmett Malcolm, Guthrie, Ky.; University of Tennessee Medical Department, Nashville, 1901; member of the American Medical Association; served during World War I; affiliated with Jennie Stuart Memorial Hospital in Hopkinsville; died Jan. 8, aged 76, of carcinoma.

Fullerton, Walter Wilson ⊕ Brockton, Mass.; College of Physicians and Surgeons, Baltimore, 1895; on the staff of Brockton Hospital; died Jan. 4, aged 80, of coronary thrombosis.

Garrick, Nathan Henry, Boston; Boston University School of Medicine, 1915; specialist certified by the American Board of Psychiatry and Neurology; member of the American Psychiatric Association; formerly assistant professor of neurology at his alma mater; on the honorary staff, Massachusetts Memorial Hospitals; died Jan. 2, aged 66, of cardiac failure.

Gear, Patrick Edward ⊕ Holyoke, Mass.; Harvard Medical School, Boston, 1919; member of the House of Delegates of the American Medical Association in 1945, 1946, and 1948; fellow of the American College of Surgeons; served as president of the staff of Holyoke Hospital and Providence Hospital, where he died Jan. 20, aged 57, of hypertensive heart disease.

Geissler, Elmer Edwin, Gloucester City, N. J.; Temple University School of Medicine, Philadelphia, 1918; member of the American Medical Association; served during World War I; medical inspector for the public school; died in West Jersey Hospital, Camden, Jan. 13, aged 64, of arteriosclerotic cardiovascular disease and Parkinson's disease.

Gibbs, Emmett Wyattman, Shelby, N. C.; University of North Carolina School of Medicine, Chapel Hill, 1907; member of the American Medical Association; died Jan. 14, aged 79, of coronary sclerosis.

Gifford, Samuel Lee, Rome, N. Y.; University of the City of New York Medical Department, New York, 1893; member of the American Medical Association; formerly practiced in Whitesboro, where he was health officer; served as health officer of the town of Whitestone; died Jan. 8, aged 82, of cerebral hemorrhage.

Grave, Floyd ⊕ Minneapolis; Johns Hopkins University School of Medicine, Baltimore, 1914; fellow of the American Society of Clinical Pathologists; past president of the Minnesota Academy of Medicine; served during World War I; affiliated with St. Barnabas Hospital, Eitel Hospital, Veterans Administration Hospital, and Asbury Hospital, where he died Dec. 20, aged 67, of coronary thrombosis.

Guagliata, Vincent, Chicago; National Medical University, Chicago, 1903; died Jan. 8, aged 75, of pyelonephritis and prostatic hypertrophy.

Hagan, Eugene Anthony, Pawtucket, R. I.; Georgetown University School of Medicine, Washington, D. C., 1931; died Jan. 30, aged 50, of coronary thrombosis.

Hamilton, James M., Chicago; Rush Medical College, Chicago, 1895; died Jan. 11, aged 84, of acute myocardial infarction.

Hasie, James LeRoy, De Kalb, Miss.; University of Tennessee College of Medicine, Memphis, 1929; member of the American Medical Association; served during World War II; affiliated with Kemper County Hospital; died in Daytona Beach, Fla., Jan. 14, aged 46, of a heart attack.

Henning, Harry Hugh, Chicago; Dearborn Medical College, Chicago, 1905; died Jan. 18, aged 80, of coronary occlusion.

Hewitt, James Ernest, Summit, Miss. (licensed in Mississippi in 1907); died Nov. 15, aged 70, of bronchopneumonia and pancreatitis.

Hubbard, William Eugene, North Little Rock, Ark.; University of Oklahoma School of Medicine, Oklahoma City, 1943; member of the American Medical Association and the Oklahoma State Medical Association; served during World War II; resident in psychiatry at Veterans Administration Hospital; died Jan. 9, aged 35, of acute coronary occlusion.

Karr, George Muster, El Paso, Texas; Jefferson Medical College of Philadelphia, 1894; died Nov. 9, aged 76.

Kestle, Charles William, San Francisco; University of Colorado School of Medicine, Denver, 1931; member of the American Medical Association and Colorado State Medical Society; died in Stanford University Hospital Dec. 14, aged 44, of carcinoma of the tongue.

Koessler, George Leo ⊕ Detroit; Detroit Homeopathic College, 1905; served on the staffs of William Booth Memorial and Grace hospitals; died Feb. 20, aged 72, of coronary thrombosis and hypertension.

Krenning, William George, St. Louis; Washington University School of Medicine, St. Louis, 1901; member of the American Medical Association; died Jan. 8, aged 76, of coronary disease.

Krueger, Bernard ⊕ Milwaukee; Wisconsin College of Physicians and Surgeons, Milwaukee, 1905; formerly practiced in Cudahy, where he was health commissioner and school physician; on the staff of St. Luke's Hospital; died Jan. 6, aged 70, of coronary sclerosis.

Ledyard, Cory Childs, Los Angeles; University of Southern California School of Medicine, Los Angeles, 1906; for many years on the staff of California Hospital, where he died Jan. 1, aged 68, of carcinoma of the prostate.

Lee, Willie Allen, North Little Rock, Ark.; College of Physicians and Surgeons, Keokuk, Iowa, 1893; died Jan. 5, aged 91.

Lewis, Henry Harrison, Buffalo; University of Buffalo School of Medicine, 1918; affiliated with Millard Fillmore Hospital, Columbus Hospital, and Deaconess Hospital, where he died Jan. 15, aged 57, of brain tumor and diabetes mellitus.

Lima, Joseph J. T., Twerton, R. I.; Baltimore Medical College, 1908; served as president of the public library; died Jan. 2, aged 79, of carcinoma of the liver.

Lincoln, Jennings Sipe, Montclair, N. J.; Johns Hopkins University School of Medicine, Baltimore, 1918; past president of Associated Physicians of Montclair and Vicinity; served overseas during World War I; affiliated with Mountainside Hospital, where he was staff president and where he died Jan. 21, aged 59, of coronary thrombosis.

Littlefield, Anna Maria ⊕ New London, N. H.; Woman's Medical College of Pennsylvania, Philadelphia, 1895; died Jan 3, aged 92, of cerebral arteriosclerosis.

Longwell, Benjamin James, Seminole, Pa.; Ohio Medical University, Columbus, 1904; member of the American Medical Association; died in Brookville (Pa.) Hospital Feb. 18, aged 72, of a fracture of the right hip sustained in a fall and colitis.

Loose, David Nathaniel, Maquoketa, Iowa; University of Pennsylvania Department of Medicine, Philadelphia, 1877; member of the American Medical Association; died in Orlando, Fla., Jan. 30, aged 96, of coronary thrombosis and diabetes mellitus.

McClain, William Zid, Marlow, Okla.; College of Physicians and Surgeons, Dallas, Texas, 1908; died at Crockett, Texas, Jan. 16, aged 68.

McClenahan, Henry Eugene ⊕ Youngstown, Ohio; University of Michigan Medical School, Ann Arbor, 1915; specialist certified by the American Board of Obstetrics and Gynecology; member of the Central Association of Obstetricians and Gynecologists; fellow of the American College of Surgeons; served during World War I; on the staff of Youngstown Hospital; died Jan. 5, aged 67, of carcinoma.

McDonald, David Lorenzo, Duchesne, Utah; Medico-Chirurgical College of Philadelphia, 1907; at one time practiced in Salt Lake City, where he was affiliated with Dr. W. H. Groves Latter-Day Saints Hospital; died Jan. 15, aged 78, of pneumonia.

McGillian, Eugene Francis, New York City; Albany (N. Y.) Medical College, 1909; attached to the U. S. Veterans Administration at its New York City regional office; served during World War I; at one time practiced in Yonkers, where he was health commissioner from June, 1937, to August, 1946, and where he was director of Gray Oaks Hospital, and on the staffs of St. Joseph's, St. John's Riverside, and Yonkers Professional hospitals; died Jan. 10, aged 65, of coronary thrombosis.

May, Harold Cameron, Los Angeles; Hahnemann Medical College of the Pacific, San Francisco, 1908; member of the American Medical Association; served during World War I; died Jan. 29, aged 68.

Meek, Henry G., Akron, Ohio; Baltimore University School of Medicine, 1893; died Feb. 6, aged 83, of coronary thrombosis.

Molotchick, Maxwell Benjamin, Lynn, Mass.; Middlesex College of Medicine and Surgery, Cambridge, 1928; member of the American Medical Association; affiliated with Union Hospital; died in City Hospital, Boston, Jan. 15, aged 48.

Moore, William Thomas, Marshall, Ark. (licensed in Arkansas in 1905); member of the American Medical Association; died in St. Vincent's Infirmary, Little Rock, Dec. 23, aged 75, of hepatitis.

Mullins, Glenn R. ⊕ Dublin, Ga.; Emory University School of Medicine, 1915; served during World War II; affiliated with Veterans Administration Hospital; died Nov. 4, aged 59, of dissecting aneurysm of the abdominal aorta.

Neas, Ingall Howard, Kansas City, Kan.; University of Kansas School of Medicine, Kansas City, 1935; member of the American Medical Association; served during World War II; died Dec. 25, aged 43.

Netzel, Charles Frederick ⊕ Plymouth, Pa.; Jefferson Medical College of Philadelphia, 1916; school physician; affiliated with Nesbitt Memorial Hospital, where he died Jan. 17, aged 57, of ruptured aneurysm of the abdominal aorta.

Nickson, Delbert Harry ⊕ Seattle; University of Oregon Medical School, Portland, 1917; specialist certified by the American Board of Pathology; member of the American Society of Clinical Pathologists; for many years affiliated with Swedish Hospital; died Jan. 2, aged 61, of rheumatic and hypertensive heart disease.

Noble, Herbert T., Bayside, N. Y.; New York Homeopathic Medical College and Hospital, New York, 1895; died Dec. 3, aged 81, of heart disease.

Norstedt, Johann Albert, Nanticoke, Pa.; University of Pennsylvania Department of Medicine, Philadelphia, 1903; served on the staff of the Nanticoke State Hospital; died in Hackensack, N. J., Jan. 20, aged 66, of cerebral hemorrhage.

O'Brien, Francis Edward, Leedy, Mass.; Fordham University School of Medicine, New York, 1917; received the Navy Cross for service during World War I; retired superintendent of the Hampshire County Sanatorium in Haydenville; died in the U. S. Veterans Hospital, Northampton, Feb. 15, aged 56, of coronary thrombosis.

Olson, William Carolius, Chicago; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1899; affiliated with Edgewater and American hospitals; died in Cook County Hospital Jan. 5, aged 76, of bronchopneumonia, following fracture of the left femur sustained in an automobile accident.

Openshaw, James Francis, Goodland, Ind.; Indiana University School of Medicine, Indianapolis, 1936; served during World War II; died in the Robert W. Long Hospital, Indianapolis, Jan. 23, aged 44, of acute toxic hepatitis.

Panetti, Philip A., Hustisford, Wis.; Kentucky School of Medicine, Louisville, 1892; member of the American Medical Association; died Dec. 16, aged 79, of bronchopneumonia and chronic myocarditis.

Price, Enoch Cyril ⊕ Lubbock, Texas; Southwestern University Medical College, Dallas, 1910; served during World War II; affiliated with Veterans Administration; died recently, aged 66.

Quick, Ralph Andre, Arlington, Va.; George Washington University School of Medicine, Washington, D. C., 1908; served with the American Red Cross in Europe during World War I; on the staff of Arlington Hospital; died Jan. 23, aged 69, of coronary thrombosis.

Reale, Frank Paul ⊕ Plainfield, N. J.; Loyola University School of Medicine, Chicago, 1938; affiliated with St. Peter's General Hospital in New Brunswick and Muhlenberg Hospital; died Feb. 7, aged 43, of rheumatic heart disease.

Reilly, Henry M. A., New York; Jefferson Medical College of Philadelphia, 1896; at one time affiliated with All Souls Hospital in Morristown, N. J., attached to the Frances Schervier Home and Hospital, where he died Nov. 6, aged 79, of cerebral hemorrhage and arteriosclerosis.

Reis, Roy Clifford, San Francisco; University of Louisville (Ky.) School of Medicine, 1928; died Jan. 5, aged 53, of coronary occlusion and arteriosclerosis.

Ripton, James Raymond ⊕ Cleveland; Western Reserve University School of Medicine, Cleveland, 1920; fellow of the American College of Surgeons; affiliated with St. John's Hospital; died Jan. 27, aged 56, of coronary thrombosis.

Rizzo, Patrick Charles, Mount Vernon, N. Y.; New York Homeopathic Medical College and Flower Hospital, New York, 1930; member of the American Medical Association; on the staff of Mount Vernon Hospital; died Jan. 15, aged 48, of acute coronary occlusion.

Rodenbaugh, Norman Fredrick, Barberton, Ohio; Ohio Medical University, Columbus, 1893; at one time health officer; formerly affiliated with Citizens Hospital; served on the board of education; died Jan. 3, aged 86.

Rosenberg, Arthur ⊕ Brooklyn; University of Maryland School of Medicine and College of Physicians and Surgeons, Baltimore, 1933; affiliated with Israel Zion, Coney Island, and Madison Park hospitals; died in Mount Sinai Hospital, New York, Jan. 4, aged 45.

Russell, John J., Putnam, Conn.; New York Homeopathic Medical College and Hospital, New York, 1887; member of the American Medical Association; for many years medical examiner of Putnam; on the staff of the Day Kimball Hospital; died Jan. 28, aged 85, of arteriosclerosis.

Ryder, Anna L. Burkhart, Chambersburg, Pa.; the Hahnemann Medical College and Hospital, Chicago, 1893; past president of the Franklin County Medical Society; on the staff of Chambersburg Hospital; died Jan. 1, aged 88, of terminal pneumonia.

Salzer, Moses ⊕ Cincinnati; Medical College of Ohio, Cincinnati, 1902; specialist certified by the American Board of Internal Medicine; assistant professor of medicine at University of Cincinnati College of Medicine; consultant in medicine, Jewish Hospital, and president of the medical staff from 1943 to 1945; died Jan. 6, aged 70, of cancer.

Saunders, Archibald McGregor, Cleveland; Detroit College of Medicine, 1897; died in Lakeland Jan. 29, aged 83, of diabetes mellitus.

Scofield, Charles J., Chicago; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1901; affiliated with Illinois Central Hospital, died in Odell, Ill., Feb. 9, aged 73, of cancer.

Scott, Martin Bruce, Delaware, Okla. (licensed in Oklahoma under the Act of 1908); died Jan. 5, aged 72, of chronic myocarditis and arteriosclerosis.

Seifert, Francis Robert ⊕ Elkins Park, Pa.; Medico-Chirurgical College of Philadelphia, 1903; affiliated with Stetson and Episcopal hospitals in Philadelphia; died Jan. 6, aged 75, of carcinoma of the prostate.

Shallcross, William G. ⊕ Pittsburgh; Jefferson Medical College of Philadelphia, 1897; on the staff of St. Margaret's Memorial Hospital, where he died Jan. 2, aged 84, of coronary occlusion, following an operation for carcinoma of colon.

Simmons, Clarence Wright, Philadelphia; Hahnemann Medical College and Hospital of Philadelphia, 1896; served for many years as a diagnostician for the bureau of health; died Dec. 29, aged 84.

CORRECTION

Physician Not Dead.—The announcement in THE JOURNAL, March 22, page 1046, of the death of Dr. Joseph Boudinot Johnston Jr., of Kannapolis, N. C., was an error. Dr. Johnston is alive and in good health. It was Joseph Boudinot Johnston Sr., a doctor of divinity, who died.

GOVERNMENT SERVICES

PUBLIC HEALTH SERVICE

Annual Report of Public Health Service.—According to the annual report of the Public Health Service, the lowest death rate in the nation's history was recorded in 1951. The health status of the average American continues to be strengthened, according to the report. The death rate for 1951 was 9.6 per 1,000 population, 10% lower than in 1940. The average life expectancy at birth has risen to 68 years, compared with 47 at the turn of the century. The infant and maternal mortality figures dropped to the lowest point ever recorded in the United States. Infant mortality has fallen from 100 per 1,000 live births in 1915 to an estimated 20 per 1,000 in 1950. The death rate from tuberculosis dropped to 22 per 100,000 population, about 50% lower than the rate 10 years ago. Less syphilis was reported in 1950 than in any year since 1929; and less gonorrhea than at any time since 1942. The 33,209 cases of poliomyelitis in 1950 represented a 21% decrease over the all-time high of 1949. Only 42 cases of smallpox were reported during the year. With the increased numbers of persons living longer, the chronic diseases, however, are taking a greater toll. Diseases of the heart and cancer caused two-thirds of all deaths in 1950. Only half of the 3,070 counties of the United States are served by public health departments. Another 550 counties receive advisory and supervisory services from some sixty state health districts.

During 1951 state programs emphasized prevention and control of chronic diseases. Control programs for cancer and heart disease received particular attention. Research in chronic disease kept pace with the expansion of control measures. Progress is reported in cancer research, in mental health research, and in dental research. To promote more extensive research, two new institutes were established during the year, the Institute of Arthritis and Metabolic Diseases and the Institute of Neurology and Blindness. Construction of the new clinical center building was reported 59% completed at the close of 1951. The center, which will be ready for occupancy in 1953, will give the National Institutes of Health, the research arm of the service, its first opportunity to conduct clinical research on a wide scale.

The Public Health Service is greatly concerned with health problems related to the national emergency. Among projects undertaken in this area during the past year were development of a plan for the rapid detection and reporting of disease epidemics, study of the effects of radioactive wastes on water uses, and special training in radiological health. The service has also responded to increasing demands for advice and counsel on health matters, for the loan of experts for defense activities, and for the staffing and conduct of special projects.

The advances toward longer and healthier American lives have been brought about by medical research, expanded hospital facilities, and improved local health services, the report said. During the past year all 53 states and territories surveyed their hospital resources and reported that there are now slightly more than one million hospital beds available. There was an increase of 142,000 beds during the year, half of them constructed with assistance under the Hill-Burton program administered by the Public Health Service.

Survey of Cancer in Pittsburgh.—The incidence of cancer among residents in the Pittsburgh area apparently increased 30% from 1937 to 1947, according to a survey just released by the National Cancer Institute of the Public Health Service. The greatest increase noted was for cancer of the bronchus and lung, the incidence for which almost doubled during the 10-year period. Some of the apparent increase in cancer incidence is attributed to improvements in diagnostic techniques and case-finding methods. Another reason is that the proportion of older persons in the population has increased. Cancer occurs 120 times as frequently in persons over 65 as in children under 15, the survey points out. The Pittsburgh survey was carried out in cooperation with the Allegheny County Medical Society, the Hospital Council of Western Pennsylvania, and city and state health departments.

The director of the Cancer Institute, Dr. John R. Heller, said, "There is evidence which suggests that cancer patients in the Pittsburgh area are receiving better medical care than they did in 1937. The survey notes that more cancer patients received hospital attention in 1947 than 10 years previously. Further, we note that greater numbers of cancers were confirmed by microscopic examination of the tissues involved. Improvements such as this have made for the positive identification of malignancies."

As in the other surveys already published, there was a much greater increase in cancer incidence among males than in females. One of the main sex differences noted in the 10-year period was for cancer of the bronchus and lung, which remained the same for females and increased 147% among males. Among males in the Pittsburgh area, the commonest cancer sites were digestive system, 36% of all cases; skin, 13%; respiratory tract, 12%; and genital organs, 11%. Among females, the commonest cancer sites were digestive system, 27%; genital organs, 23%; breast, 20%; and skin, 9%.

The Pittsburgh survey indicates that early diagnosis is closely related to chances for survival. This was clearly shown by the 12-month survival rates for cancer of the uterus. When diagnosed in a localized stage, the 12-month survival rate for uterine cancer was 89%. When there was regional involvement, 75% of the women with uterine cancer survived for one year. This percentage dropped to 15% when cancer diagnosis was made after metastasis had set in.

Close Hospital on Martha's Vineyard.—The Public Health Service Hospital at Vineyard Haven has discontinued admitting patients and will be closed as soon as provision can be made for patients now in the hospital. Surgeon General Scheele recommended closing the 30 bed federal hospital because the number of patients treated has declined to about 40% of its capacity. Dr. Scheele based his decision on the findings of a special survey board appointed to study the operations of the hospital. The board noted that only 29% of the patients admitted to the hospital in recent periods were residents of Martha's Vineyard.

The Public Health Service will assume full medical care for coast guardsmen and seamen, who are the principal Service beneficiaries at the Vineyard Haven Hospital. The Public Health Service will appoint a private physician on Martha's Vineyard to render outpatient care to eligible persons. Patients requiring hospitalization will be referred to the Public Health Service Hospital in Boston, a 300 bed institution with comprehensive medical, surgical, and allied professional services. The new Veterans Administration hospital recently opened at Providence, R. I., and others soon to be completed in the Boston vicinity provide amply for veteran patients now served by the Vineyard Haven Public Health Service Hospital.

MISCELLANEOUS

Emergency Portable Laboratory.—A portable clinical laboratory chest for use in improvised hospitals during emergencies has been developed by Federal Civil Defense Administration's Health and Welfare Office. The wooden frame chest, two ft. long, about that high, and a foot deep, when completely filled weighs about 75 lb. The portable laboratory is built to provide blood and urine tests, and some other procedures, such as spinal fluid, fecal specimen, and sputum examinations. Built according to FCDA's requirements, these chests will be part of the equipment stored in federal warehouses, available to the states in emergencies immediately following enemy attack. The chest, fully equipped, costs about $125. Pilot models for trial are being purchased in small quantities. Procurement of larger quantities may be possible next year if federal funds are available.

FOREIGN LETTERS

BELGIUM

Gastric Diseases and Diseases of Blood.—At a meeting of the Belgian Society of Gastroenterology, several authors studied the aspects, repercussions, and relationships of diseases of the blood and those of the digestive tract. Lambin gave a résumé of the opinions expressed in the course of this discussion.

It is useful to investigate the secretion of hydrochloric acid, not only in patients with pernicious anemia but also in those with chronic hypochromic anemia, with the injection first of 1 mg. of histamine and afterwards of 4 mg. Patients who react to this last dose have only a transient case of achlorhydria; the stomachs of the others have lost the power of secretion. Patients with pernicious anemia are, with rare exceptions, refractory to histamine. It is important in hypochromic anemia to prescribe hydrochloric acid simultaneously with trivalent preparations of iron to assure its absorption or else to give bivalent iron.

The digestive manifestation most characteristic of iron deficiency is the Plummer-Vinson syndrome, which responds rapidly to appropriate treatment with iron. The characteristic modifications may be evidenced by a suitable radiological technique (Laurell). Gastric observations in the course of pernicious anemia show that gastritis is frequent but not constant and that its development is independent of that of anemia. Patients with pernicious anemia are predisposed to gastric polyposis and to cancer of the stomach. The gastrogenic theory of polycythemia rests on a slender foundation, but the increased incidence of ulcers among patients with this disease has not yet received a satisfactory explanation. Esophageal and gastroduodenal lesions are rare in the course of leukemias. The most typical is that of chronic lymphadenosis.

Digestive localizations of Hodgkin's disease are comparatively rare; primary lesions are exceptional and are generally mistaken for cancer. A diagnosis of cancer is likewise usually made in cases of lymphosarcoma and reticulosarcoma. Extirpation, in all these cases, may lead to a long survival. Irradiation treatments are indicated because of the radiosensitivity of lymphogranulomatous and lymphosarcomatous or reticulosarcomatous tissues, in spite of the inconveniences arising from the resolution of the visceral lesions, which leads to the production of fibromatous cicatrices and perivisceritis.

In the hemorrhagic diatheses mention must be made of gastric localization of familial hemorrhagic angiomatosis. The hemorrhages of this condition are favorably influenced by large doses of rutin. Isolated gastric purpura deserves more ample study.

The massive digestive hemorrhages that often complicate fibrous and congestive enlargement of the spleen (Banti's syndrome) are not explained simply by the rupture of esophageal varices. Account must be taken of the alterations in the veins of the submucosa in the areas tributary to the splenic vein as well as neurogenic vasomotor attacks. The frequency of gastroduodenal ulcers in these patients is likewise unexplained. The problem of the surgical treatment of this affection is being fully studied. Not only must splenectomy be carried out but the associated disturbances of the portal circulation must also be corrected.

The search for the cause of secondary anemia of digestive origin has made notable progress, thanks to the increasing precision of radiological techniques. The importance of diaphragmatic hernia and especially that of the esophageal hiatus in these conditions has been demonstrated by radioscopy.

Prolapse of the gastric mucosa through the pylorus must also be noted as a cause of anemia. Finally, stress must be laid on the useful results of iron treatment in the hypochromic anemias of gastric cancers. Macrocytic anemia is less frequent, and true pernicious anemia is rare in cases of cancer.

The anemia observed after extensive gastrectomy is only one of the minor inconveniences of this operation. Pernicious anemia, in fact, is rare and slow to appear. Hematological observation will reduce the incidence of iron-deficiency anemia, and the administration of easily absorbable iron will rapidly relieve it.

Histidine and Diagnosis of Pregnancy.—Before the Royal Belgian Society of Gynecology and Obstetrics, Cheval and Hans reported the following clinical results obtained from the examination of urine for histidine in 175 patients, for the purpose of establishing a diagnosis of pregnancy.

1. The presence of histidine in the urine makes it possible to affirm that if a woman does not menstruate in the three days following the examination she is pregnant.

2. The absence of histidine in the urine shows that a woman is not pregnant or has a pregnancy that is not developing normally.

3. The amount of histidine in the normal woman remains stable throughout pregnancy.

4. The reduction in the amount of histidine in the course of pregnancy is pathological. In some cases, there are reasons to anticipate a toxemia of pregnancy or an infection of the urinary tract (colibacillus).

In the course of the discussion, Dr. Cheval pointed out to those who had previously obtained less convincing results that it was not formerly known that histidinuria exists in all women at the time of menstruation. He stressed the necessity, moreover, of using fresh reagents for the detection of histidine and also the fact that he had obtained a positive result in a case of fibroma in a woman who was not pregnant. De Rom, on his part, drew the attention of the assembly to the existence of positive histidinuria in almost all patients with epithelioma of the cervix.

Council of State Annuls Code of Ethics.—The decision of the Council of State to annul the Code of Medical Ethics has given rise to profound repercussions in medical circles and has awakened a lively curiosity on the part of the general public. The code, which was destined to serve as a guide, a kind of vade mecum for the Belgian physician, did not pretend to be either perfect or immutable. Because it did not meet the requirements of a government law, it has been annulled. But the decision will not alter the respect the majority of physicians have for it. It is certain that the present development of medicine requires important adjustments in all fields; the whole problem will have to be taken up again for the protection of the medical fraternity and in the interest of patients themselves.

Teaching of Industrial Medicine.—The Faculty of Medicine of Louvain, in collaboration with the Superior Industrial Institute, which has just been created at the university, is organizing, beginning with this year, a postgraduate course of instruction in industrial medicine, ending with the granting of a diploma as licentiate in industrial medicine.

The program includes the following subjects: biometry and physiology of labor; labor hygiene; occupational diseases; industrial toxicology; industrial traumatology, invalidism, and readjustment; industrial psychology; technology of industrial safety and hygiene; organization of work and personnel; social medicine; and labor legislation.

Creation of an Institute of Hygiene and Epidemiology.—The *Moniteur Belge* announces the creation, within the Ministry of Public Health and of the family, of an Institute of Hygiene and Epidemiology, combining the central laboratory of hygiene and bacteriology, the laboratory of inspection of foodstuffs, the laboratory of inspection of pharmacies, the laboratory of inspection of trade in meats, the office of vaccinations, and the sanitary inspection of ports, airports, and frontiers.

The items in these letters are contributed by regular correspondents in the various foreign countries.

ITALY

Italo-Swiss Convention.—Many specialists attended the Italo-Swiss convention of otologists, neurosurgeons, and ophthalmologists, held in Stresa, under the chairmanship of Professors Franceschetti and Montandon from Geneva. Professor Simonetta read a paper on the modern treatment of cerebral abscess. During the discussion that followed, neurosurgeons and otologists reached opposite conclusions, the former supporting radical treatment and the latter, conservative treatment. The neurosurgeons were in favor of a massive exeresis, and Professor Fasiani, a clinical surgeon from Milan, discussed 60 cases, showing roentgenograms, arteriograms, and encephalograms.

On the other hand, the otologists treated cerebral abscesses of otogenous origin with punctures, drainages, and antibiotics. Professor Casedesus, an otologist, stressed the importance of the use of a contrast medium in the abscess cavity, so that its growth could be followed. From the standpoint of mortality, the statistics given by both neurosurgeons and otologists were almost equal; however, it is difficult to compare treatment of, and death rate caused by, cerebral abscesses of different origin.

Professor Chaussé, from Paris, read a paper on tumors of the jugular foramen, stressing hemorrhagic tumors. These tumors often appear with a lesion of the seventh cranial nerve, and their growth lasts some years. Warning of the tumor's presence is given by otologic signs; roentgenographic investigations have great importance in diagnosis. Neurological signs are scarce, because the tumor develops posteriorly and does not compress the organs in the jugular foramen. Later, a bleeding polypus of the ear appears, inducing the patient to consult an otologist.

Professors Montandon and Monnier, from Geneva, described the simultaneous recording of nystagmus and changes in arterial pressure obtained by rotary excitation of the vestibular apparatus. Little study has been done on the action of rotary stimulation on the arterial pressure; the speakers, in their effort to define the characteristics of pressure variations during the prerotary and postrotary phases of stimulation, performed experiments on lightly anesthetized experimental animals.

On the basis of the clinical material from the optical clinic in Lausanne and his own material, Professor Streiff, from Lausanne, reported on trauma of the optical pathways. Speaking of trauma of the optic nerve, he emphasized the importance of the condition of the pupils, of stereoroentgenography, and of the pathogenetic causes of amblyopia and blindness. Treatment consists of decompression intervention on the optic canal. Traumatic lesions of the optic chiasm, the acoustic striae, and the retinal cortex are rarer. Professor Franceschetti, from Geneva, is in favor of opening the sphenoid sinuses in those instances in which the general condition of the patient does not justify a decompression operation on the optic canal. One patient who suffered cranial trauma regained his sight after the speaker opened the sphenoid sinuses. Professor Franceschetti advocated retrobulbar injections of vasodilator substances in patients with severe cases and also as postoperative adjuvants. Prof. Sanvenero Roselli, from Milan, discussed plastic operations of the orbit. He is against the use of plastic substances; he uses a fragment of bone taken from the patient's iliac crest.

Arteriography with Gaseous Contrast Medium. — Professor D'Errico reported to the Neapolitan Surgical Society the experimental and clinical results of his method of performing arteriography with a gaseous contrast medium, oxygen. The speaker thought of this method after he considered that oxygen is a good and perfectly harmless contrast medium. The danger of gas emboli forming after injection of oxygen into the arteries is practically nonexistent, because the gas spreads into the capillary bed, where part of it is absorbed by the tissues and part of it combines with hemoglobin of the blood before it reaches the right ventricle through the venous system. Professor D'Errico performed the first gaseous arteriographies in man; he injected, with a syringe, by means of manual pressure, small quantities (50 to 60 cc.) of oxygen into the femoral artery, using a very thin needle connected to the oxygen-filled syringe by means of a rubber tube. The results were satisfactory in that it was possible to visualize in all instances the artery into which the oxygen had been injected and its main branches.

Pneumoarteriography compared to arteriography with opaque contrast medium has the following advantages. 1. The contrast medium is completely harmless, so that the injection may be repeated more than once at the same sitting. 2. It is possible to use a very thin needle for the injection. 3. There are no arterial spasms; thus, the arteriograms correspond to the real condition of the artery. 4. There is no danger of damage from an accidental periarterial injection.

Although the arteriograms are not so clear as those obtained when an opaque contrast medium is used, Professor D'Errico found that they are always sufficient for the diagnosis.

LONDON

The Cost of Health.—In a book that was just published, "The Cost of Health," Dr. Ffrangcon Roberts, a radiologist, who is equally well known for his frank writings on health problems and medical education, has made a candid analysis of the National Health Service. His book has provoked widespread interest, in both the lay and the medical press. Dr. Roberts is concerned with the reasons why the National Health Service, which, according to the National Health Service Bill, of 1946, was estimated to cost £152,000,000 a year, should now be costing over £400,000,000 yearly. In spite of this tremendous increase in expenditure, physicians' offices are crowded, hospital waiting lists are lengthening rapidly, and promised health centers are now almost figments of the imagination. Dr. Roberts does not believe that the increased cost of living, abuse of the service by patients, or administrative extravagance are responsible for more than a fraction of this increase. His diagnosis suggests a more deep-seated cause. In his analysis, he starts with the simple proposition that "the amount of treatment which any nation can afford to provide depends on productive capacity."

He points out that "it has been the advance of medicine which has set the pace throughout." In 1860, there were only 11,000 physicians in England and Wales, and, assuming their average income to be £500, the total, £5,500,000, represents practically the entire national expenditure on medical services. Today, there are over 45,000 medical practitioners in England and Wales. In 1900, there were 19,000 hospital beds in England and Wales, and, in 1946, there were over 90,000. "The increasingly rapid demand for medical treatment is the expression, not of a belated attempt to make up for past neglect, but of the efforts of the community . . . to keep up with the results of scientific research." The rate of progress has risen by leaps and bounds, and, in the last 10 years, it has been greater than ever before: "In these years medicine has undergone little short of a revolution, it is advancing more rapidly than the capacity of statesmen and administrators to deal with it." Figures are quoted from one general hospital, showing an increase in x-ray examinations from 516 in 1918, to 19,000 in 1950, with the cost of each examination in 1947, being three times as expensive as in 1930. There was an increase in medical examinations from 1,500 in 1927, to 39,260 in 1950, with a 32% increase during the last year, and there was an increase in the waiting list from 220 in 1937, to 2,700 in 1949. The increase in the cost per inpatient per week rose from £1.12*s.* in 1906, to £8.4*s.* in 1947, and the increase in the cost per outpatient per attendance rose from 1*s.*4*d.* in 1927, to 5*s.* in 1947. In 50 years, the number of beds in this hospital has been doubled, but the total staff has increased to 7 times the original number, the medical staff 12 times, and the nursing staff 5 times. This increasing acceleration in cost is not confined to England and Wales, and "the miscalculation of the cost of the Service was primarily due to the complete failure on the part of the Ministry of Health to realize that the system which the State took over in 1948 so far from being static, was intensely and increasingly dynamic."

In discussing "the mutual obligations of the individual and the state," it is pointed out that "it is the universal experience that state provision for sickness is followed by an increase in the incidence of sickness, and there is no evidence that the National Health Service is reversing this trend." Evidence from the United States and prewar Germany as well as from England and Wales is cited in favor of this conclusion. For instance, in the first year of the National Health Service, there was a rise in the absence rate in the Royal Ordnance Factories from 2.6 to 5.9%. Further, "there is no law to prevent patients from using their disabilities to tyrannise their fellow-men. They can abuse the services of their doctors . . . By their legal powers they compel general practitioners and hospitals to take troublesome protective

measures." Then there is the paradox of the State providing both infertility and birth control clinics: "There is grim humour to be derived from the contemplation of a community, the vast majority of which voluntarily reduces its effective fertility, attempting to restore the balance by subsidising medicated procreation in the minority not naturally endowed with the capacity."

In comparing private and state insurance, Dr. Roberts points out that "in private insurance voluntary self-denial is an essential element. The insured foregoes the pleasures of the moment for security in the future, and is fully conscious of it. . . . In national health insurance the reverse is true; the burden of the compulsory contributions has been put forward as a reason for higher wages. . . . Private insurance companies honor their undertakings or go out of business. How the State has defaulted needs no emphasis . . . From the very outset the Government admitted that it could not meet its obligations . . . Private companies take elaborate precautions to ensure that there is no pecuniary advantage in being ill. . . . In State insurance, on the other hand, there are many ways in which it pays to be ill . . . In all forms of private insurance the one essential is limitation of benefits . . . The promise of unlimited benefits, which is an essential part of the Service, cannot be honoured."

The chapter on the economic value of the physician can best be summarized by the following quote: "What is to count in medicine in the future? The qualities and experience which should result from a medical training, or the test-tube and the machine? Is medicine to degenerate into mass-production in which technicians multiply and leadership lies in the hands of those members of the profession with a flair for organisation and committee-work? If so, it is obvious that the State cannot at the same time continue to pay a number of four-figure salaries which would drive a commercial concern to bankruptcy. . . . If the doctor is to retain his ascendancy and his claim to a high salary he must use the machine only as an adjunct to his own brain. If he uses it instead of his brain the State cannot be expected to pay for both . . . There is subtle irony in the fact that, whereas the establishment of medicine as a profession was accompanied by the suppression of unqualified practice, the advance of medicine which has resulted is the Trojan horse which admits unqualified practice in the respectable form of lay assistance."

His summing up is epitomized in the following quotation: "A free and comprehensive health service is a noble ideal, but one which the country cannot under existing conditions attain . . . When the people realise that the benefits which can be derived from the Service depend primarily on national production, and that these benefits are adversely affected by all inflationary tendencies—restrictive practices, strikes, lock-outs, go-slow tactics—then only will they be worthy of the fruits of their own inspiration."

Scottish Air Ambulance Service.—In the highlands and islands of Scotland, transportation and communication facilities are inevitably primitive compared with the rest of the country, and, during the long northern winters, many of these Scottish islands are stormbound. The medical service provided for them by the Department of Health for Scotland has long been recognized as a model of its kind; one of its features is its air ambulance service. This service was inaugurated in 1933, when a physician on the island of Islay arranged for a desperately ill patient to be flown to hospital in Glasgow. The service developed rapidly, and, after the 1939 to 1945 war, responsibility for it was taken over by British European Airways. Rapid biplanes are used, three seats being removed to make room for a stretcher. A plane is always available for emrgency calls, and the service attempts to have the plane in the air within half an hour of receiving a call during the day and within an hour at night. During the 19 years since the service was established, there has never been a fatal accident, an outstanding record in view of the poor flying conditions and the inadequate landing grounds. As often as not, the islands are stormbound or cloudbound, and, in some instances, the landing ground is a 500 yard grass strip or a stretch of sand. The latter may be under water during high tide.

Any physician in an outlying area has authority to send for an air ambulance if he has a case that he considers sufficiently urgent to require immediate removal to the hospital. This is done by telephoning movement control at the appropriate airfield near Glasgow. The rest is done by British European Airways. A nurse flies in each air ambulance, and a pool of nurses for this purpose is provided by one of the Glasgow hospitals. As the plane approaches the airfield at Glasgow or Aberdeen, it notifies movement control of its expected time of arrival, and an ambulance is waiting on its arrival to take the patient to the hospital. This service is frequently used; the busiest year was 1949, during which aircraft flew over 72,000 miles in carrying 350 patients on 305 flights. Most of the cases, in their order of frequency, are surgical, medical, accident, or maternity. The youngest patient was a 3 lb. (1.3 kg.) premature infant, if one omits the infant born on the aircraft on its way to Glasgow, and the oldest patient was a lady of 90. When helicopters become available, it is hoped that it will be possible to make use of them in providing an air ambulance service to those smaller islands where fixed wing aircraft are unable to land.

SPAIN

Congress on Digestive Diseases and Nutrition.—The Sixth Congress of Pathology of the Digestive Tract and of Nutrition was recently held in Santander. Mr. Ruiz Jiménez, Minister of National Education, was president. Dr. A. Cortés Lladó, professor of surgery of the faculty of medicine of Seville, spoke on surgical treatment of cancer of the colon. In the beginning, cancer of the colon is relatively benign; early diagnosis and treatment are important. To find the initial site from which the tumor is growing as well as to ascertain its size and relation to nearby structures, a thorough examination of the colon and a careful interpretation of the clinical symptoms are necessary. Adequate preoperative and postoperative treatment will result in lowering the mortality rate to 5% and in prolonging the life span of the patients from three to five years after the operation. The patients should have antibiotics and other supportive treatment. Cancer of the colon calls for surgical removal of the involved segment. The incision for the operation should be large, permitting visualization of the entire abdominal cavity, so that the extent and stage of the disease can be judged. When a complete operation cannot be performed because of acute obstruction, acute infection, adhesions, or the patient's low resistance, operations for internal or external derivation are indicated. External derivation is preferable when the tumor is in the left colon. Care is taken that the maneuvers of mobilizing and lowering the colon do not interfere with the anus. There should be extensive resection of the intestinal segment and its mesentery, including certain lymph nodes and zones of adhesion. The arteries and veins should be carefully observed to determine which are to be preserved and which should be sacrificed. The remaining part of the intestine should have a sufficient blood supply. Colectomy with immediate reconstruction of the intestine is advisable, regardless which segment of the colon the tumor is located in, if the tumor is not infected and the patient has complete preoperative treatment. Lateral anastomoses are useful when there is fatty infiltration of the intestines and the blood supply of the intestinal ends is poor. In left colectomy, a colostomy is useful. In the patients treated by the speaker, the mortality rate varied between 5 and 10%. The life span after the operation was more than five years in 60% of the patients.

Prof. A. Gallart Esquerdo of Barcelona discussed the diagnosis of cancer of the colon, as based on personal experience. He observed 18 cases of cancer of the cecum, 21 of the ascending segment of the colon, 8 of the hepatic angle, 10 of the transverse segment of the colon, 1 of the splenic angle, 11 of the descending segment of the colon, and 31 of the sigmoid flexure. There are no rigid criteria regarding age and sex of the patients; psychogenic trauma is a causal factor. In 16.6% of the cases observed by the speaker, cancer of the cecum involved the ileocecal valve (histopathological verification). The speaker classified cancer of the colon into two macroscopic types, the infiltrating type and the ulcerovegetating cancer. With scirrhous cancer, there are no indications of its histological type. Any type of cancer may develop in either the right or the left segments of the colon; however, the ulcerovegetating form predominates in the right colon and the infiltrating form in the left colon. Polypi were observed in 4% of the patients. The degree of malignancy of the tumor should be determined by histological study of representative sections. In the cases observed by the speaker, per-

foration into the bladder, the skin, and the free peritoneum did not occur. Colon-duodenal, colon-gastric, and colon-jejunal fistulas and involvement of the kidney or the genital organs of the women were observed. There is no direct relationship either between the apparent origin of cancer of the colon and the involvement of the lymph nodes or between the involvement of the lymph nodes and the local growth of the tumor. Metastases to the lymph nodes occur late. The clinical symptoms of early cancer of the colon are varied. Any symptom suggesting a diseased intestinal condition may correspond to the early stage of the disease. The classical general symptoms (precordial anorexia, etc.) appear so late in the course of the disease that, sometimes, the cancer is in an advanced stage before any symptoms are detected. When an intestinal tumor becomes palpable, the cancer has already reached an advanced stage, often with complications. The speaker described, from a didactic viewpoint, two clinical forms of cancer that occur in either the right or the left colon. The diagnosis of cancer of the colon is made by rectosigmoidoscopy, together with vaginal and rectal digital palpation and roentgen examination. The roentgen examination is preceded by insufflation of air. Serial radiographs are taken in the course of the introduction of barium and especially of thorium. These examinations should be repeated periodically until there is an image of the stricture or remote circular dilatation. These images are the most frequently observed in the advanced stages of cancer; however, the changes that exist in the proximal and distal zones of the normal colon, which are in contact with the neoplastic segment, constitute the most valuable data for the diagnosis. Early diagnosis of cancer of the colon is extremely rare at present. The layman does not recognize the early symptoms of the disease as they are mild and of long duration. Nearby or remote metastases often appear when the symptoms have lasted only a few months. Furthermore, physicians often think of cancer of the colon only when the size of the tumor is already noticeable.

The third topic discussed was the syndrome after cholecystectomy. Dr. S. Carro, of Madrid, was the official speaker. The diagnosis is based on the results of a thorough examination. Among the most frequent operative complications after cholecystectomy are the following: lithiasis of the common bile duct, stricture of Oddi's sphincter, pancreatitis, perivisceritis, and extrabiliary diseases, some of which may have been present before the operation and may have been overlooked during it. The name of syndrome after cholecystectomy should be reserved to designate the disorders caused by changes of the vesicular functions but not other diseases that may have been either overlooked or diagnosed erroneously. Some of these sequels or syndromes, such as the residual lithiasis of the common bile duct, the stricture of the bile ducts or Oddi's sphincter, perivisceritis, biliary fistula, and stenosing pancreatitis, call for surgical treatment. Medical therapy is indicated in dyskinesia due to inflammation, infection, endocrine or psychogenic disorders, and functional changes caused by the removal of the gallbladder.

M. Martin Lagos, professor of surgery of the faculty of medicine of the University of Madrid, did not agree with the name given to the disease that may appear after cholecystectomy; he believed that it is no more than a recurrence of the symptoms that were present before the operation. The treatment consists in preventing the recurrence during the first operation. Removal of the gallbladder results in diminishing true recurrences.

SWEDEN

Infectious Hepatitis as a Medical Risk.—In Sweden and Denmark, steps have been taken to ascertain whether physicians are more likely to develop infectious hepatitis than other members of the community. In Sweden, Dr. J. P. Strömbeck has organized a statistical enquiry among physicians to whom a questionnaire was addressed on this subject. More than 4,000 members of the Swedish Medical Association who received this questionnaire were classified into three groups according to whether they were (1) in general practice, (2) in hospital practice not concerned with operations (including physicians and radiologists), or (3) in hospital practice requiring operations (including surgeons and obstetricians). Among the 4,030 recipients of this questionnaire, there were 3,802 who answered it satisfactorily. There were 531 physicians who, at some time, had suffered from hepatitis once and 22 who had hepatitis more than once. In many instances, hepatitis had developed early in life, and special interest was attached to the patients in whom this disease had appeared after the age of 25. The hepatitis rate was higher among physicians in hospitals than among those in general practice. It was also higher among the hospital physicians who operated than among those who did not operate; the difference between these two groups of physicians was not great enough to be statistically significant.

In Denmark, Dr. Sten Madsen addressed a similar questionnaire to some 4,700 physicians, 94% of whom sent in answers. The answer rate was almost exactly the same as that in Sweden, which was 94.3%. Dr. Madsen found that about 8% of the physicians questioned had suffered from hepatitis before the age of 18 years and that about 10% had done so in adult life. In Denmark, as in Sweden, it seems that, in adult life, the risk of developing infectious hepatitis is greater for physicians than for others. To provide himself with satisfactory control material, Dr. Madsen is planning to investigate the hepatitis rate in other groups in the community, such as lawyers.

According to the survey taken in Sweden, it may be noted that since infectious hepatitis was included among the notifiable diseases in 1931, its frequency rate has risen enormously. During the period 1931-1937, the annual rate was about 1,300. During the period 1943-1946, there were some 7,000 cases notified every year, and, in 1948, the notifications rose to 11,736. It is particularly in the large towns that recent epidemics of hepatitis have assumed disquieting proportions, and Stockholm's fever hospital has had to organize a special department for the accommodation of some 100 patients at a time. A clear distinction is now drawn between the epidemic hepatitis resulting from infection of the digestive tract (with an incubation period of from three to five weeks) and inoculation hepatitis (with an incubation period of two to four months). The serious problems raised by both these forms of hepatitis led the Swedish ministry of health, in 1949, to issue a memorandum on the subject.

Payment for Blood Donors.—Dr. Bengt Gullbring, who is a member of the staff of Caroline Hospital (Karolinska Sjukhuset) in Stockholm, has reviewed, in the organ of the Swedish Medical Association, *Svenska Läkartidningen,* the pros and cons of giving blood donors material compensation for their services. It is a common experience that the supply of blood given gratis requires many appeals, whereas much less publicity is needed for the recruiting of donors given substantial payment for their blood. In Sweden, it is customary to pay a donor 20 to 30 crowns out of public funds for each donation. In 1949, 1,500,000 crowns were paid for such services.

In the spring of 1951, Dr. Gullbring sent a questionnaire to 1,000 donors, who were requested to express their opinions on payment or nonpayment for their blood. The names of 748 of the donors were protected by anonymity. The first question was; "What is the most important reason for which you give blood? Is it out of idealism, i. e., to help ailing fellow-beings, or for profit?" The answers showed that idealism was the chief motive in 18%, profit the chief motive in 52%, and both motives in 15%; no answers or obscure answers were given in the remaining 15%.

The second question was, "Would you give blood if you were not paid for it in cash (28 crowns)?" The answer was in the affirmative in 34%, and in the negative in 46%; the donor did not know in 15%. No answers or obscure answers were given in the remaining 5%. Several of the donors who were unwilling under present conditions to give their blood gratis wrote that they would do so in case of war. It should be noted that the recipients of this questionnaire were already donors on a commercial basis. They could not, therefore, necessarily be regarded as representative of the community as a whole. For this and other reasons, Dr. Gullbring warns against the application of his findings to the question of payment or nonpayment as a whole. Nor can conditions in Sweden be compared directly with those in other countries, such as the United States, where the donor is paid by the patient directly and is not reimbursed out of public funds. But it may be taken as a general rule that gratuitous blood donation presupposes repeated public appeals to idealism. Such appeals must be wisely spaced. Otherwise, an injudicious rush of appeals may yield an embarrassing rush of overgenerous donors.

CORRESPONDENCE

ANESTHESIA IN OPERATION FOR PHEOCHROMOCYTOMA

To the Editor:—In a paper concerning anesthesia in operations for pheochromocytoma, in THE JOURNAL, Jan. 19, 1952, Papper and Cahill state, "no ill-effects from the sympathomimetic action of ether have been noted." They also state that the use of N, N-dibenzyl-beta-chloroethylamine (dibenamine®) 24 hours preoperatively "is not without risk, because of a sustained hypotension."

Decker and associates (*J. A. M. A.* **147:**642 [Oct. 13] 1951), in discussing the operative treatment of a case of pheochromocytoma, mention a fatality due to irreversible shock following removal of the tumor and suggest that "dibenamine® apparently had rendered the patient refractory to epinephrine."

Recently in January, 1952, a case of paroxysmal hypertension in a 31-year-old white man was successfully cured with the operative removal of a pheochromocytoma by Dr. John B. Flick. The anesthetic problems in this patient were the most difficult to manage. Thiopental (pentothal®) sodium was used for induction, but sufficient relaxation was not obtained to permit endotracheal intubation. Ether was given and the tube inserted. Despite administration of 2-(N-*p*-tolyl-N-[*m*-hydroxyphenyl]-aminoethyl)-imidazoline hydrochloride (regitine®) intramuscularly just before induction, and despite the continuous use of piperoxan (benodaine®) intravenously, the diastolic pressure rose to 160 mm. Hg, with a systolic pressure controlled by the above-mentioned drugs at between 200 and 220 mm. Hg. The diastolic pressure was not controlled until a spinal anesthetic, hypobaric dibucaine (nupercaine®) was administered. It then dropped to between 110 and 120 mm. Hg.

The pressure variations secondary to manipulation of the tumor and its removal were easily controlled by the continuous use of intravenous drip solutions of benodaine® and norepinephrine (levophed®). Postoperatively, the norepinephrine solution was necessary to support the blood pressure for approximately 36 hours. Thus the combination of spinal anesthesia with the short acting adrenergic blocking agents seemed successful in controlling the paroxysms of hypertension. Preoperatively, the tumor was successfully stimulated by exercise, histamine, and methacholine (mecholyl®) bromide. The first two procedures were successful in breaking through the relative hypotension induced by regitine.® It would seem probable that ether in this case acted as a dangerous stimulant or provocative agent to the tumor in breaking through the control by regitine® and benodaine.® These facts seem of sufficient importance to warrant early communication.

EDWARD W. BIXBY JR., M.D.
MARIO V. TRONCELLITI, M.D.
The Pennsylvania Hospital and Benjamin Franklin Clinic, Philadelphia.

COARCTATION OF THE AORTA

To the Editor:—The interesting report by Dr. Raymond A. Sokolov, of Detroit (*J. A. M. A.* **148:**641-642 [Feb. 23] 1952) of a case of coarctation of the aorta with surgical correction at age 40, the case of a man aged 41 successfully surgically treated by Dr. Blalock (Gross, R. E.: *J. A. M. A.* **139:**285 [Jan. 29] 1949), and the first successful case reported by C. Crafoord and G. Nylin (*J. Thoracic Surg.* **14:**347, 1945) lead me to mention at this time the early cases reported by G. B. Morgagni (De sedibus et causis, 1761) and Dr. Paris (*J. de chir. de Desault* **2:**107-110, 1791), whose female patient was about 52 years old. Discovery of a markedly coarctated aorta was demonstrated at the time he was injecting the body for dissection, and Laennec (1781-1826), who had seen several cases, referred to four other cases of marked obliteration of the aorta recorded in the literature by Ashly Cooper, John Bell, and Graham (Mediate Auscultation, translated by Forbes, ed. 3, 1832, p. 686). However, the oldest proved case of (nearly) complete obliteration of the aorta recorded in medical literature, with illustration of the "specimen" (diagrammatic), I found to be the case of a man aged 92 years, reported by Dr. A. Reynaud in 1828, nearly 125 years ago (Observation d'une oblitération presque complète de l'aorte, *J. hebd. de méd., Par.* **1:**161-175, 1828). Craigie in 1841 collected 10 cases; Peacock in 1866 collected 46 cases. It is of further interest that L. M. Bonnet in 1903 analyzed a total of 160 cases. It was Bonnet who divided coarctated aortas into (1) the infantile or developmental type with usually a diffuse narrowing at the isthmus and (2) the adult type with a sudden constriction at the site of the ductus, due to postnatal cicatricial contraction (*Rev. de méd., Par.*, **23:***108, 1903).* Corvisart, Leroux, and Boyer recorded a case of coarctation of the aorta in a man past 40 years (*J. de méd., chir., pharm., etc., Par.*, **33**, 1815). Finally, in 1827 Meckel, perhaps for the first time, showed rib erosion in a case of coarctation and rupture of the right auricle. This observation has been confirmed by x-ray studies by Hugo Rösler (1928), now of Philadelphia (*Wien. Arch. f. inn. Med.* **15:**521-538, 1928). In this article, rib erosion in one case was given passing notice. Railsback and Dock (*Radiology* **12:**58-61, 1929) and many others since 1929 have also confirmed this observation. Jordan (1830), Craigie (1841), Walshe (1873), and Byrom Bramwell (Diseases of the Heart and Thoracic Aorta, Edinburgh, 1884) observed erosion of the ribs. D. Evan Bedford (at a meeting of the Cardiac Club in 1930) and W. Dock (Erosion of Ribs in Coarctation of Aorta: Note on History of Pathognomonic Sign, *Brit. Heart J.* **10:**148-149, 1948) observed rib erosion with coarctation of the aorta. In 1835, Legrand clinically diagnosed obstruction of the thoracic aorta; and Oppolzer, in 1848, made probably the first clinical diagnosis of stenosis of the aorta at the ductus (Hamernjk, cited by Flexner, J.: Coarctation of Aorta [Adult Type]: Clinical and Experimental Studies, *Am. Heart J.* **11:**572-580, 1936).

HYMAN I. GOLDSTEIN, M.D.
1425 Broadway, Camden 4, N. J.

WARFARIN FOR SUICIDE

To the Editor:—I read with interest the article "Suicide Attempt with Warfarin, a Bishydroxycoumarin-Like Rodenticide," which appeared in the March 15, 1952, issue of THE JOURNAL. The authors were Lt. Roy W. Holmes and Capt. Julian Lowe.

I would like to report a somewhat similar experience that I had in February, 1952, as follows. I was called to the residence of J. L. H., a young white man. The story was that he had become despondent, because his young bride of a few weeks had left him, and he went to a drugstore and asked for "rat poison." He was sold a package of "warfarat," made by Dr. Hess & Clark, Inc., Ashland, Ohio. The label on the package indicated that it contained bishydroxycoumarin (dicumarol®) and gave instructions as to the antidote. The patient stated that he took a "heaping tablespoonful" in a glass of water about one hour before his family called me. When I arrived, he had no complaints and the physical examination was essentially negative. His prothrombin time and activity were within the normal range. I had him drink several large glasses of warm soda water, which caused copious vomiting. I then gave him twelve 5 grain (0.32 gm.) tablets of vitamin K at one dose and instructed him to take two such tablets every four hours for the next 24 hours. The next day, he still had no complaints; his prothrombin time and activity were again normal, and I dismissed him.

I agree with the authors of the above-quoted article that warfarin is an excellent rat poison, but that a one-dose episode would not be lethal for a human.

J. MORGAN KELLUM, M.D.
Silvertown Clinic, Thomaston, Ga.

BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Chiropractic: Applicant Must Obtain Preliminary Education In Proper Sequence.—The petitioner filed a mandamus action to compel the medical licensing board of West Virginia to permit him to take the examination for a license to practice chiropractic in the state. The case was heard by the Supreme Court of Appeals of West Virginia.

The West Virginia statutes provide: "Any person wishing to practice chiropractic in this State shall apply to the secretary of the medical licensing board for a license so to practice. Each applicant shall be a graduate of a chiropractic school or college recognized by the American Chiropractic Association, or other recognized national chiropractic society, which teaches a resident course of at least three calendar years of eight months each and requires active attendance upon the same, and shall be a graduate of an accredited high school giving a four-year course or have an education equivalent to the same, and shall have attended for at least two years an academic college equal in standing to the West Virginia University, as preliminary education."

The petitioner attended Marshall College at Huntington, West Virginia, during the academic years 1944-1945 and 1945-1946 and received 46½ hours college credit; he then received an additional 8 hours college credit for the time he served in the United States Navy; he then attended and was graduated from a chiropractic college recognized by the American Chiropractic Association; thereafter, during the academic year 1950-1951 he again attended Marshall College and received an additional credit of 15 hours. Upon applying to the medical licensing board for permission to take the examination for a license to practice chiropractic, the petitioner was advised that he was not eligible to take the examination for the reason that the 15 hours' credit obtained at Marshall College during the academic year 1950-1951, was after he had received his chiropractic training.

It appeared that there has been an unbroken administrative interpretation of long standing by the chiropractic board of examiners and its successor, the present medical licensing board, interpreting the West Virginia law concerning the qualifications prerequisite to the taking of the chiropractic examination to mean that an applicant must show that he has completed at least two years of academic college work prior to and as preliminary education to taking the graduate course at a chiropractic school or college; that this long established interpretation by the board is "consonant with the public policy in this state and the high standards necessary to protect the public requiring applicants for examinations for particular professions to first complete a minimum of undergraduate or academic college training or high school training as preliminary training prior to taking graduate training in a particular professional school. . . ." The basic question in this case, said the Supreme Court of Appeals, is whether a writ of mandamus should issue to compel the medical licensing board to allow the petitioner to take the examination in view of the fact that he had the necessary high school, college, and professional credits, although a necessary part of his academic college credits were obtained after his graduation from a chiropractic college.

From a careful consideration of the language employed in the statute, said the court, we are of opinion that graduation from high school or the equivalent thereof, and attendance at an academic college equal in standing to West Virgnia University must precede attendance at a chiropractic school or college. In reaching this decision, we are supported to a large extent by the fact that there has been an unbroken administrative interpretation of long standing by the chiropractic board of examiners and its successor, the medical licensing board, that the statute provides that a high school and academic college education shall be had before attendance at and graduation from an accredited chiropractic school. "A contemporary exposition of a statute, uncertain in its meaning, recognized and acquiesced in for a long period of time by the officers charged with the duty of enforcing it, the courts, the Legislature, and the people, will be adopted unless it is manifestly wrong." We hold, the court concluded, that the medical licensing board, in refusing the petitioner admission to the examination for a license to practice chiropractic on the basis of its interpretation of the statute, was correct. Accordingly the petition for a writ of mandamus was denied. *State ex rel Ballard v. Vest, 65 S. E. (2d) 649 (West Virginia 1951).*

Medical Practice Acts: Revocation of License; Advertising.—The State Board of Medical Examiners revoked the defendant's license for unprofessional conduct and the defendant appealed to the Supreme Court of Oklahoma.

The Oklahoma statute permits the revocation of a physician's license for "all advertising of medical business in which statements are made which are grossly untrue or improbable and calculated to mislead the public." The advertisement used by the defendant contained the following: "Complete Examination including Fluoroscopic of the Respiratory System, Mouth, Throat, Sinuses, Heart, Spinal and Nervous System, Abdomen, Kidneys, Pelvis and Rectum, Blood Pressure and Urinalysis Test. Hemorrhoids (Piles) Treated Tonsils Removed. . . ."

In construing a similar provision in a prior law relating to the revocation of a physician's license for unprofessional conduct, the Supreme Court of Oklahoma said ". . . As we regard this section of our statute, we think that it is not aimed at any unethical practices of physicians as interpreted by the medical fraternity, but was aimed to prevent acts on the part of physicians which are universally regarded as immoral and against good conscience, not only by the medical profession, but by laymen as well, and for which under the style of obtaining money under false pretenses our Criminal Code has provided the penalties of the law."

The only witness produced on the question of the use of the word "fluoroscopic" was a specialist in the field of radiology. He testified that while the fluoroscope was useful in the examination of some, if not most, of the organs or parts of the body listed in the advertisement, it was of little or no assistance in examining the kidneys, and of no assistance in examination of the mouth. He admitted that the advertisement was ambiguous and that he was unable to determine to what extent it represented that the fluoroscope would be used. He testified that in the examination of some of the other organs or parts of the body listed, such as the spinal and nervous systems and the abdomen, the fluoroscope had a limited field. The whole extent of the radiologist's testimony was to establish that its use, as to some of the organs or parts of the body listed, was of no practical value in examining them. No witness testified to having been misled by the advertisement, nor did any witness testify that the fluoroscope could not be used in the examination of the various organs.

It is to be noted, said the Supreme Court, that the advertisement nowhere asserts that the fluoroscopic examination of the various organs mentioned therein is superior to any other form of examination, or gives any assurance of any extraordinary results to be produced thereby, nor did the evidence show that any of the statements were untrue, or that by reason thereof any members of the public had been, or probably would be, misled to their injury, and we are unable to say from an inspection of the advertisement that it would necessarily have such effect. The Supreme Court concluded that while the advertisement may be unethical practice from the viewpoint of the medical fraternity, it does not violate the statute according to the construction placed upon it in the prior decision. Accordingly the order revoking the defendant's license was reversed. *Choate v. State, 232 P. (2d) 634 (Oklahoma 1951).*

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Am. Journal Diseases Child., Chicago

83:1-150 (Jan.) 1952

Use of Wetting Agents in Treatment of Respiratory Infections of Infants. G. C. Hall Jr.—p. 1.
Importance of Rest in Treatment of Early Convalescent Poliomyelitis. W. Green.—p. 4.
Prophylactic Immunization and Poliomyelitis. J. Lieben.—p. 8.
*Fanconi Syndrome: Multiple Congenital Anomalies with Hypoplastic Anemia. H. K. Silver, W. C. Blair and C. H. Kempe.—p. 14.
*Gamma Globulin Studies in Tuberculosis in Children. B. M. Levin, H. Kaufman and J. de la Huerga.—p. 26.
Community Study of Rheumatic Fever. H. A. Joos and C. P. Katsampes. —p. 37.
Factors to Be Considered in Planning a Premature Center. H. M. Wallace, M. A. Losty and L. Baumgartner.—p. 54.

Fanconi's Syndrome.—Fanconi's syndrome is an unusual combination of disorders that includes severe progressive refractory macrocytic anemia with pancytopenia (anemia, neutropenia, and thrombocytopenia), a hypoplastic bone marrow, and a generalized brown, melanin-like pigmentation of the skin, associated with various congenital defects. The defects often include microcephaly, bone abnormalities (particularly of the forearms and thumbs), hypogenitalism, genitourinary tract abnormalities, strabismus, and in at least one case, unilateral deafness, gynecomastia, and congenital heart disease. Fanconi first described this syndrome in 1927 in three brothers, each of whom died of an aplastic-type of anemia. Silver and associates report a case of Fanconi's syndrome in a girl, first admitted to their hospital in January, 1949, at the age of 11. She had anemia, thrombocytopenia, congenital abnormalities of the skeletal system, microcephaly, microphthalmos, a generalized olive-brown pigmentation of the skin, and enlarged breasts. The girl was hospitalized many times. Whole blood transfusions proved to be the only effective means of therapy, but were only of temporary value, and splenectomy was without apparent benefit. The occurrence of leukocytosis under the stimulus of multiple staphylococcic infections of face, buttocks, cervical nodes, and lungs was of particular interest. The girl died in November, 1950. The observations at necropsy are described in detail. The pathogenesis of the hematological changes observed is not entirely clear. The hypoplastic marrow and pancytopenia are similar to those found in aplastic anemia. However, the elevated reticulocyte count seen early in many cases of Fanconi's syndrome, and the marked anisocytosis and poikilocytosis, which have usually been noted, differ from the picture of normal red cells and decreased reticulocyte count usually seen in cases of aplastic anemia of either the primary idiopathic or the secondary type. Serial bone marrow examinations suggest that the inability of the marrow to produce red cells is progressive. This is substantiated by the concomitant reduction in the number of reticulocytes in the peripheral blood.

Gamma Globulin Studies in Tuberculous Children.—Antibodies for most diseases, including tuberculosis, have been found in the gamma globulin fraction. A recently described turbidimetric method for the determination of the gamma globulin produces results that correspond closely to results of the more complex electrophoretic determinations. Using this quick method serum gamma globulin levels were determined weekly for from three to eight months in 73 children with various types of tuberculosis to ascertain whether or not the levels followed any particular pattern during the course of tuberculosis. Sedimentation rates, hematocrit readings, and total and differential white cell counts were determined concurrently to see what relationships, if any, there are among results of these tests in tuberculosis. The gamma globulin level was greater than normal levels fairly early in tuberculosis. In uncomplicated primary pulmonary tuberculosis and in tuberculosis with effusion, peak elevations of gamma globulin were reached at about the sixth week, with return to normal values by about the 20th week. This paralleled the course of the sedimentation rate, but the latter rose a little earlier and returned to normal earlier. In reinfection tuberculosis the high levels of gamma globulin were maintained longer, probably owing to the fact that the disease remained active longer in these cases. The highest levels were maintained for the longest periods in tuberculous adenitis. This agrees with the theory that gamma globulin is a product of the reticuloendothelial system, as lymph nodes are made up largely of reticuloendothelial elements. Patients with bone tuberculosis had the lowest gamma globulin levels. In cases of miliary tuberculosis without meningitis the levels were very high. This may indicate a good antibody response, and may explain how these patients avoided meningitis. Patients with tuberculous meningitis had initial rises in gamma globulin, but there was no correlation between the gamma globulin and erythrocyte sedimentation rates just before the onset of the meningitis and just before death, when the gamma globulin level fell rapidly. The serum gamma globulin level, when followed serially and in conjunction with the sedimentation rate, is a valuable index of prognosis. A falling gamma globulin occurring with a rising sedimentation rate is a bad prognostic sign, indicating decreased body resistance and/or increased disease activity. A single gamma globulin determination is of no prognostic significance.

A.M.A. Arch. Dermat. and Syphilol., Chicago

65:1-122 (Jan.) 1952

Boundaries of Dermatology: Presidential Address. H. E. Michelson.—p. 1.
Pregnancy and Apocrine Gland Diseases: Hidradenitis, Fox-Fordyce Disease. T. Cornbleet.—p. 12.
Dermatology in Egypt. G. R. Mikhail and M. S. Falk.—p. 20.
Clinical Study of "Quotane": New Antipruritic Drug. F. W. Lynch and O. E. Ockuly.—p. 35.
*Intravenous Use of Procaine Hydrochloride in Control of Pruritus. L. G. Beinhauer, G. J. Thomas and S. R. Perrin.—p. 39.
Circle Strobe Lighting in Medical Photography. D. J. McNairy and S. R. Mercer.—p. 45.
Pigmented Nevus of Fingernail Matrix: Report of Case. J. F. Noble, A. Ferrin and K. A. Merandino.—p. 49.
Keloid Resulting from Positive Patch Test. J. E. Dalton.—p. 53.
Treatment of Exfoliative Dermatitis with Cortisone. B. M. West.—p. 56.
Lepra Reaction: Its Treatment with Dihydrostreptomycin. B. Saenz. —p. 59.
Serum Lipoproteins and Cholesterol Metabolism in Xanthelasma. N. N. Epstein, R. H. Rosenman and J. W. Gofman.—p. 70.
Koebner Phenomenon in Lichen Nitidus: Report of Two Cases. H. Pinkus and H. M. Shair.—p. 82.
Xanthoma Disseminatum: Unusual form with Extension of Xanthomatous Changes into Muscle. S. B. Frank and A. I. Weidman.—p. 88.

Procaine Hydrochloride Given Intravenously in Pruritus.—Procaine hydrochloride was given intravenously to 181 patients with pruritus. The majority had been treated with other methods without being relieved. A dose of 500 cc. of a 0.1 to 0.2% solution of procaine hydrochloride in isotonic sodium chloride solution was best tolerated when given over a period of 60 to 90 minutes. Barbiturate medication was given one hour before injection, and 200 mg. of ascorbic acid was added to each infusion to increase the resistance against toxic side-effects and to benefit patients with poor nutrition. When edema was present, 5% dextrose was added. In patients with nervous irritability and fear of the infusion thiopental (pentothal®) sodium in doses of 200 to

Periodicals on file in the Library of the American Medical Association may be borrowed by members of the Association or its student organization and by individual subscribers, provided they reside in continental United States or Canada. Requests for periodicals should be addressed "Library, American Medical Association." Periodical files cover only the last 11 years, and no photoduplication services are available. No charge is made to members, but the fee for other borrowers is 15 cents in stamps for each item. Only three periodicals may be borrowed at one time, and they must not be kept longer than five days. Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted.

300 mg. added to the first two or three injections relieved the pruritus and allowed the patient to sleep. This modified treatment proved particularly effective in generalized neurodermatitis. Most patients were given one injection daily, and it was learned that, if pruritus was not relieved within six days, further therapy was generally useless. Most patients who reacted favorably were relieved after the fourth intravenous injection. The procaine hydrochloride injections produced complete relief in 59 patients, temporary relief in 67 patients, and no relief in 55 patients. When properly controlled, the treatment is safe, and can be tried when other therapies fail. The following are contraindications for this therapy: allergy to procaine, myasthenia gravis, thyrotoxicosis, and treatment with digitalis or digitalis-like drugs.

A.M.A. Arch. Indust. Hyg. & Occup. Med., Chicago

5:1-96 (Jan.) 1952

*Treatment of Chronic Pulmonary Disease with Intermittent Positive Pressure Breathing: I. Evaluation by Objective Physiological Measurements. H. L. Motley and J. F. Tomashefski.—p. 1.
Tetryl Toxicity: Summary of 10 Years' Experience. B. B. Bergman.—p. 10.
Exhaust Ventilation for Machine Tools Used on Materials of High Toxicity. H. F. Schulte, E. C. Hyatt and F. S. Smith Jr.—p. 21.
Further Investigations on Chronic Cadmium Poisoning: Study on Rabbits with Radioactive Cadmium. L. Friberg.—p. 30.
Condensation Plastics: Their Dermatological and Chemical Aspects. G. E. Morris.—p. 37.
Effects of Dimethyl and Diethyl Paranitrophenyl Thiophosphate on Experimental Animals. W. B. Deichmann, W. Pugliese and J. Cassidy.—p. 44.
Physiological Response of Experimental Animals to Vapor of 2-Nitropropane. J. F. Treon and F. R. Dutra.—p. 52.
Spiral Sampler—New Tool for Studying Particulate Matter. C. D. Yaffe, A. D. Hosey and J. T. Chambers Jr.—p. 62.
Management of Older Employee with Medical Problems. R. B. Crain.—p. 71.

Intermittent Positive Pressure Breathing in Chronic Pulmonary Disease.—In 300 cases of silicosis maximal breathing capacity and vital capacity measurements were taken before and immediately after one intermittent positive pressure treatment combined with the simultaneous administration of a bronchodilator, a synthetic racemic epinephrine (vaponefrin®) hydrochloride solution, using a specially designed therapy unit. In 41 cases of silicosis similar determinations were made before and immediately after one treatment, and after a course of treatments given at the rate of three a day for two to three weeks. The number of treatments, the duration of single treatments, and the quantity of racemic epinephrine used were varied for each patient. All degrees of bronchial obstruction and decrease in lung elasticity were observed. In general the obstructive pattern was pronounced, but in most cases the increase in maximal breathing capacity noted after the treatment was interpreted as being due to relief of bronchospasm. Electrical recordings of instantaneous flow rates were made during maximal and deep breathing before and after one intermittent positive pressure breathing and bronchodilator treatment in one patient with asthma, one patient with chronic bronchitis, three patients with silicosis, and one patient with diffuse fibrosis following irradiation treatment. Tracings provided useful information concerning bronchospasm and elasticity changes in the lungs. The improved bronchial drainage, the more uniform distribution of aerosol to all parts of the lungs with consequent more effective relief of bronchospasm, and the improved muscle tone resulting from the breathing exercise are the most important results of this treatment, which appears promising in providing relief and control of symptoms in emphysema and fibrosis with dyspnea. It should be emphasized that this is not a cure in the sense that the normal condition of the lung is restored, but most of the patients were able to increase their activities after treatments. Maximal breathing capacity is the most significant and simplest objective measurement that can be correlated with subjective and clinical improvement.

Delaware State Medical Journal, Wilmington

23:353-384 (Dec.) 1951

Acute Toxemias of Pregnancy. K. Vileisis.—p. 353.
Reactive Depression: Case Report. H. W. Geyer.—p. 355.
Artificial Triceps Action. G. J. Boines.—p. 357.

Journal of Bacteriology, Baltimore

62:677-810 (Dec.) 1951. **Partial Index**

Isolation of Cryptococcus Neoformans from Soil. C. W. Emmons.—p. 685.
Activators for Cysteine Desulfhydrase System of Escherichia Coli Mutant. E. A. Delwiche.—p. 717.
Further Evidence of Existence of Mitochondria in Bacteria. S Mudd, A. F. Brodie, L. C. Winterscheid and others.—p. 729.
Process of Hyaluronidase Formation by Hemolytic Streptococci. B. Sallman.—p. 741.
Observations on Action of Citrovorum Factor in Leuconostoc Citrovorum. Sing Chen Chang, M. Silverman and J. C. Keresztesy.—p. 753.
Minimal Nutritional Requirements of Lactobacillus Bifidus. J. B. Hassinen, G. T. Durbin, R. M. Tomarelli and F. W. Bernhart.—p. 771.
Inhibition by Nitrate of Enzyme Formation During Growth of Escherichia Coli. D. Billen.—p. 793.

Journal of Immunology, Baltimore

67:449-566 (Dec.) 1951

Attempts at Quantitative Estimation of Second Component of Complement. M. Heidelberger, J. Jonsen, B. H. Waksman and W. Manski.—p. 449.
Serological Response of Cotton Rats and Monkeys to Poliomyelitis Virus. M. Pollard.—p. 463.
Antiviral Properties of Ehrlichin, Antibiotic Produced by Streptomyces Lavendulae. V. Groupé, J. W. Frankel, M. P. Lechevalier and S. A. Waksman.—p. 471.
Protection of Mice Against Vaccinia Virus by Administration of Phenoxythiouracils. R. L. Thompson, M. Price, S. A. Minton Jr. and others.—p. 483.
Zone of Localization of Antibodies: XIII. In Vivo Localization of Anti-Liver-Blood-Vessel Antibodies in Rat. D. Pressman, B. Sherman and L. Korngold.—p. 493.
Antigen—Antibody Reactions in Agar: II. Elementary Theory and Determination of Diffusion Coefficients of Antigen. E. L. Becker, J. Munoz, C. Lapresle and L. J. Le Beau.—p. 501.
Interaction of Influenza Virus and Intact Human Erythrocytes Observed by Replica Technique in the Electron Microscope. M. Rhian, A. S. Evans and J. L. Melnick.—p. 513.
Electron Microscopy of Chick Embryo Membranes Infected with Newcastle Disease. L. Kilham, C. Morgan and R. W. G. Wyckoff.—p. 523.
Occurrence of Newcastle Disease Antihemagglutinin in Human Sera and Its Relationship to Mumps Virus. A. S. Evans.—p. 529.
Micro-Test for Measuring Circulating Antibodies Against Virus (PVM). J. M. Smith and G. S. Mirick.—p. 539.
Concurrent Infection with Influenza A and B Viruses in Single Epidemic of Influenza. E. D. Kilbourne, H. C. Anderson and F. L. Horsfall Jr.—p. 547.

J. of National Malaria Society, Columbia, S. C.

10:287-374 (Dec.) 1951. **Partial Index**

Studies on Anopheline Larvae: I. Anatomy and Function of So-Called "Notched Organs" of Nuttall and Shipley on Thorax of Larvae of Anopheles Quadrimaculatus. S. L. Chang and F. E. Richart Jr.—p. 287.
Decline and Last Recorded Outbreaks of Malaria in North Carolina. H. F. Schoof and D. F. Ashton.—p. 306.
Duration of Untreated or Inadequately Treated Plasmodium Falciparum Infections in Human Host. D. E. Eyles and M. D. Young.—p. 327.
Observations on Gametocyteless Strain of Plasmodium Falciparum. G. M. Jeffery.—p. 337.
Promising DDT-Synergist Combinations for Control of Resistant Flies. W. T. Sumerford, R. W. Fay, M. B. Goette and A. M. Allred.—p. 345.

Journal of Thoracic Surgery, St. Louis

22:549-664 (Dec.) 1951

*Pneumonectomy for Severe Irradiation Damage of Lung. M. Bergmann and E. A. Graham.—p. 549.
Reconstructive Surgery of Trachea and Bronchi: Late Results with Dermal Grafts. P. W. Gebauer.—p. 568.
Myxochondroma of Trachea. B. H. Evans.—p. 585.
*Analysis of Twenty-Seven Cases of Acute Cardiac Arrest. J. L. Ehrenhaft, D. W. Eastwood and L. E. Morris.—p. 592.
Cardiostathorrhaphy—Splinting Flaccid Myocardium of Coronary Occlusion by Surgical Procedures. D. S. Allen.—p. 609.
Use of Auricular Appendage as Autogenous Myocardial Graft. B. G. P. Shafiroff, H. C. Baron and Q. Y. Kau.—p. 631.
Traumatic Bronchial Rupture with Plastic Repair. D. L. Paulson.—p. 636.

Pneumonectomy for Pulmonary Damage.—Roentgen irradiation of the human chest is almost always carried out for the treatment of a malignant neoplasm. Such neoplasms may be intrathoracic, e. g., mediastinal lymphomas, esophageal carcinoma, or inoperable bronchiogenic carcinoma, but the commonest indication is carcinoma of the breast. Patients who receive roentgen therapy for breast cancer deserve special attention, not only

because they constitute the largest single group but also because radiation in these cases is usually confined to one hemithorax and because their chances for long-term survival are better. To date the authors have performed a unilateral pneumonectomy in two patients, both of whom presented the typical clinical picture of irradiation damage of one lung. Cough and dyspnea were the chief complaints and were quite disabling. The radiographs of both patients were compatible with the diagnosis of radiation damage. In both cases pneumonectomy afforded marked relief, though in the first case empyema developed a long time after operation. The pathologic findings in these lungs are of interest because they represent the first human lungs with irradiation damage studied after surgical removal instead of post mortem. In both cases hyaline membranes, regarded as so constant in autopsy material, were absent. Since such membranes were not seen in experimental animals either, it is probable that this phenomenon is a terminal or agonal artifact. The authors believe that pneumonectomy should be employed in unilateral irradiation damage in cases in which the severity of the symptoms warrant such a procedure, and the history and examination suggest that the patient will survive for a reasonable length of time. No effective nonsurgical treatment of pulmonary irradiation damage is available.

Acute Cardiac Arrest.—Acute cardiac arrest may occur during minor surgical procedures. There exists no greater surgical emergency, and yet it is poorly understood by most surgeons. At the Iowa University Hospitals 25 instances of acute cardiac arrest occurred in approximately 71,000 anesthetic and operative procedures in a period of 10 years. Two additional cases that occurred in poliomyelitis patients are added to this group. There were no survivals among the 11 cases observed before 1947, when treatment consisted of intravenous or intracardiac injection of medications and artificial ventilation. In the second group of patients artificial circulation was employed and resulted in survival for various periods. The histories of the 16 patients in whom artificial circulation was employed are presented. The approach to the heart was transthoracic in most instances and transdiaphragmatic or transabdominal in a few cases. When cardiac arrest occurs, artificial ventilation with pure oxygen must be instituted immediately. Efforts of the surgical team must be directed toward reestablishing effective blood flow. The quickest approach by which manual compression of the heart can be effected should be used. Usually this will be a direct transthoracic approach. If, however, an abdominal incision already exists, transdiaphragmatic or transabdominal manipulation of the heart may be tried, but quickly changed to the transthoracic approach if adequate peripheral circulation does not appear. A palpable peripheral pulse and satisfactory blood pressures must be obtained. Adjuvant therapy may include the use of procaine intravenously, into the heart, or topically. This may reduce the irritability of the myocardium and danger of ventricular fibrillation. In the event that ventricular fibrillation does occur, electric shock with electrodes applied to each side of the heart should be used in an attempt to reverse the fibrillation. Epinephrine may have caused many deaths by producing ventricular fibrillation; if used, it should be given in small, very dilute quantities. A dosage of 0.1 mg. in 1:10,000 dilution has been observed to cause ventricular fibrillation. Under no circumstances should the content of the usual epinephrine ampoule be administered. If prolonged artificial circulation should be necessary, maintenance of the circulating blood volume may be necessary by intravenous or intra-arterial transfusions.

New England Journal of Medicine, Boston

245:957-996 (Dec. 20) 1951

Subglottic Membrane: Complication of Endotracheal Intubation. B. Etsten and D. Mahler.—p. 957.
Effects of Pyromen Administered Intravenously in Subpyrexial Doses to Depressed Patients: Preliminary Study. E. M. J. Fitch and A. C. Washburne.—p. 961.
Antibiotic Synergism and Antagonism. E. Jawetz, J. B. Gunnison and R. S. Speck.—p. 966.
Multiple Myeloma: Report of Four Cases Demonstrating Bence-Jones Proteinemia Found During Routine Complement-Fixation Tests. F. C. Collier, A. Reich and J. W. King.—p. 969.
ACTH and Cortisone in Treatment of Ocular Disease. R. W. Smith Jr. and E. H. Steffensen.—p. 972.

New York State Journal of Medicine, New York

51:2807-2948 (Dec. 15) 1951

Birth Trauma to Proximal Femur. G. J. Baron, N. Egel and F. N. Zuck.—p. 2859.
Cineangiocardiography as Diagnostic Aid in Congenital Heart Disease. R. S. Meltzer, J. S. Watson, E. S. Olsan and S. Weinberg.—p. 2865.
Postural Abnormalities of Spine and Lower Extremities in Young Children. M. B. Howorth.—p. 2868.
Metabolic Aspects of Steroid Therapy in Rheumatic Disease of Children. R. A. Higgons and A. A. Albanese.—p. 2875.
Iodinated Organic Compounds as Contrast Media for Radiographic Diagnoses: XIV. Influence of Pancreatic Function on Cholecystography. M. Radakovich, V. W. Logan, R. H. Greenlaw and others.—p. 2880.
Portal Hypertension. J. D. Stewart.—p. 2884.
Abdominal Trauma. F. G. Stoesser and R. E. Smith.—p. 2887.
Surgical Management of Obstructive Jaundice. C. E. Welch.—p. 2891.
Surgical Treatment of Hyperthyroidism. L. W. Sloan.—p. 2897.
New Method of Bone Plate Fixation. G. S. King.—p. 2903.
Prevention of Postspinal Headaches by Parenteral Hydration. W. M. Shaw.—p. 2905.
Mesenteric Thrombosis. S. B. Reich and L. Love.—p. 2909.
Treatment of Neurologic and Psychiatric Disorders Secondary to Arteriosclerosis: Preliminary Report. T. E. Bamford Jr.—p. 2913.

Northwest Medicine, Seattle

51:1-92 (Jan.) 1952

Acute Hospital Bed Needs. L. E. Powers, B. M. Bennett and E. L. Turner.—p. 23.
Outline of Undesirable Effects of ACTH and Cortisone. J. L. Bakke.—p. 26.
What the Physician Should Know About Vocational Rehabilitation. L. M. Farner.—p. 30.
Fatigue. H. A. Dickel, H. H. Dixon, R. A. Coen and R. D. Peterson.—p. 32.
Surgical Management of Benign Intraductal Papilloma of Breast. J. L. Vadheim.—p. 35.

Pediatrics, Springfield, Ill.

8:753-880 (Dec.) 1951

*Myositis Ossificans Progressiva. H. D. Riley Jr. and A. Christie.—p. 753.
Factors Affecting Prognosis of Tuberculous Meningitis Treated with Streptomycin. L. Finberg.—p. 768.
Hepatitis and Nephrosis Due to Soluble Bismuth. S. Karelitz and A. D. Freedman.—p. 772.
Influence of Diet on Occurrence of Hyperphosphatemia and Hypocalcemia in Newborn Infant. I. F. Gittleman and J. B. Pincus.—p. 778.
Hypervitaminosis A: Report of Unusual Case with Marked Craniotabes. J. M. Arena, P. Sarazen Jr. and G. J. Baylin.—p. 788.
Congenital Lobar Emphysema. R. Robertson and E. S. James.—p. 795.
Case of Adrenogenital Syndrome with Hypertension Treated with Cortisone. T. H. Shepard II and S. W. Clausen.—p. 805.
Serum Complement in Acute Glomerulonephritis and Nephrotic Syndrome. K. Lange, L. Slobody, F. Graig and others.—p. 814.
Crystalline Vitamin B_{12} and Sodium Folvate in Prematurity. T. Mitchell, J. N. Etteldorf, A. H. Tuttle and G. W. Clayton Jr.—p. 821.
*Left Coronary Artery from Pulmonary Artery: Three Cases, One with Cardiac Tamponade. H. I. McKinley, J. Andrews and C. A. Neill.—p. 828.

Myositis Ossificans Progressiva.—The occurrence of myositis ossificans progressiva is reported in a boy, aged 15 months, and in three girls, aged 18 months, 16 years, and 12 years. At the onset of the disease the two latter patients were 2-months-old and 7-years-old, respectively. Clinical observations in these four patients proved that at the onset in typical cases palpable masses over the fascial planes of the head and neck will usually develop. Biopsy suggests that the masses are tumor tissue, but subsequently calcification and ossification occurs. The term myositis ossificans is inaccurate, since there is calcification of the connective tissue between the muscles rather than calcification of the muscles. When the disease progresses as it did in the authors' patients, they frequently present themselves with gait or posture disturbances or with other orthopedic complaints. Microdactylism was present in all four patients and hallux valgus in two. The disease progresses from inflammation, swelling, and induration of the muscles and connective tissue to tumefaction and calcification in two to eight months. Finally, the calcification progresses to ossification and severe crippling. Serial illustrations and roentgenograms on several of the authors' patients demonstrated these progressive changes. Results of all biochemical determinations were normal. Cortisone, 10 mg. at six hour intervals, was given a therapeutic trial in the male infant. After 10 days of treatment the patient had an improved range of motion in his formerly fixed neck and arms, and began to feed himself. Two days later he was able to walk with the aid of a toddler.

Prior to therapy with cortisone the patient had not been able to sit alone and had to be fed because of the ossification in his back and axillary folds. The patient's course was followed for 10 months, and with 10 mg. of cortisone daily he maintained his improvement with no lessening of the range of motion, but a new tumor developed in the lumbar region. Cortisone was also given to the 16-year-old girl who had had the disease since the age of 2 months, but it proved ineffective. The pronounced subjective improvement following cortisone therapy in one early case suggests that possibly myositis ossificans progressiva is related to disturbed steroid metabolism.

Left Coronary Artery Arising from Pulmonary Artery.—Origin of the left coronary artery from the pulmonary artery is reported in three infants, two boys aged 7½ weeks and 19½ weeks and a girl aged 16½ weeks, making a total of 35 cases of this anomaly reported in infants. Two of the authors' patients survived only a few hours after admission to hospital, but a correct diagnosis had been made when electrocardiograms were available before opening the heart. In the third case the diagnosis was made by reviewing the roentgenographic and electrocardiographic findings and a possible surgical approach was discussed but the patient died. Since eight cases of this anomaly have been reported in adults, one of whom lived to the age of 64, it is suggested that the life of the infant might be saved if the condition can be diagnosed before irreversible myocardial changes have occurred. In one of the authors' patients the condition was complicated by cardiac rupture, a complication not previously reported. In the authors' three patients, as well as in the majority of those reported by other workers, there was a recurrent symptom pattern. At birth the infants appear normal, but at an early age begin to show the strain of nursing and refuse to suck the breast. This is usually a cause for weaning, and may not be interpreted as a sign of exertional dyspnea. Since it is easier to suck from a bottle, on weaning dyspnea frequently disappears temporarily. Later it becomes progressive until frequently it is present at rest and in many cases causes an erroneous diagnosis of pneumonia. On clinical examination, cyanosis is usually absent except terminally, and prominence of the left chest may be the only sign. Since this suggests cardiac hypertrophy, roentgenograms should be taken, which will show cardiac enlargement, and in many cases this can be recognized as left ventricular enlargement. An electrocardiogram is essential for diagnosis and shows inverted T waves in leads 1 and 2, a low voltage curve, and variable left axis deviation. The ideal treatment, if vasodilator drugs fail, would seem to be a direct anastomosis of the left coronary artery with the aorta; since this is technically impossible at present, other methods of increasing the flow to the left coronary artery should be attempted. Gasul suggests the Pott's-Smith operation, which by raising the pulmonary pressure, might increase the left coronary flow.

Psychiatry, Washington, D. C.

14:361-478 (Nov.) 1951. **Partial Index**

The Psychiatric Interview. H. S. Sullivan.—p. 361.
Sibling Rivalry and Social Heredity. J. R. Hilgard.—p. 375.
Data Concerning Certain Manifestations of Incorporation. H. F. Searles. —p. 397.
Comparison of Individual and Multiple Psychotherapy. J. Warkentin, N. L. Johnson and C. A. Whitaker.—p. 415.
Headache as Internalized Rage Reaction: Preliminary Report. S. L. Lustman.—p. 433.
Psychotherapeutic Principles in Social Research: Interdisciplinary Study of Hutterites. J. W. Eaton and R. J. Weil.—p. 439.

Quarterly J. Studies Alcohol, New Haven, Conn.

12:563-710 (Dec.) 1951

Relationship Between Deficiency of B Vitamins and Alcohol Intake in Rats. J. Mardones.—p. 563.
Treatment of Alcoholism with Tetraethylthiuram Disulfide in a State Mental Hospital: Clinical Study Based on 43 Cases. I. A. Shaw. —p. 576.
New Adjuvant in Postalcoholic Psychomotor Agitation. M. D. Kissen, H. E. Yaskin, H. F. Robertson and D. R. Morgan.—p. 587.
Court Clinic for Alcoholics: Description and Evaluation of the Stoughton Clinic. M. Brunner-Orne, F. T. Iddings and J. Rodrigues.—p. 592.
Nonaddictive Pathological Drinking Patterns of Homeless Men. R. Straus and R. G. McCarthy.—p. 601.
Occupational Performance of Thirty Alcoholic Men. H. L. Hochwald. —p. 612.

Radiology, Syracuse, N. Y.

57:797-962 (Dec.) 1951

"Single-Portal-Massive-Dose" X-Ray Therapy Technic for Certain Upper Respiratory Tract, Parotid Gland, and Recurrent Breast Cancers. M. Friedman and L. A. Davis.—p. 797.
*End-Results in Treatment of Ovarian Carcinoma with Surgery and Deep X-Ray Irradiation. H. E. Schmitz and J. T. Majewski.—p. 820.
Role of Surgery in Management of Cervical Carcinoma. H. E. Schmitz, J. E. Towne and C. J. Smith.—p. 826.
Hazard of Radiation. A. E. Heustis and D. Van Farowe.—p. 832.
*Steroids in Cancer of Breast. R. Rosh and G. G. Green.—p. 837.
Further Studies on Radiosensitivity of Analogous Mouse Mammary Tumors dbrB and C3H. A. Goldfeder.—p. 845.
Secondary Carcinoma of Phalanges. F. W. Brason, E. G. Eschner, S. Sanes and G. Milkey.—p. 864.
Primary Melanosarcoma of Esophagus. J. M. Burnett and E. St. John. —p. 868.
Roentgenologic Considerations in Tracheo-Esophageal Fistula Without Esophageal Atresia: Report of Two Cases. T. F. Leigh, O. A. Abbott and W. A. Hopkins.—p. 871.
Pantopaque Meningitis Due to Hypersensitivity. J. C. Luce, W. Leith and W. S. Burrage.—p. 878.
Adaptometer for Preparation for Fluoroscopy. F. A. Riebel and C. C. Carroll.—p. 882.
New Radiolaryngometric Method. C. E. Gárciga.—p. 884.

Ovarian Carcinoma Treated by Surgery and Irradiation.—Schmitz and Majewski analyzed the records of 143 patients with malignant ovarian neoplasms who were followed for at least five years at the Institute of Radiation Therapy at the Mercy Hospital in Chicago. Contact was lost with 13, who have been counted as deceased in the final tabulations. The onset of ovarian carcinoma is insidious. Abdominal swelling or the presence of a mass was the presenting symptom in 65%. It is estimated that three to six months will have elapsed before the most active of these growths produces recognizable abdominal enlargement, and at least 12 months before the less malignant tumors cause enlargement. By this time extension to the peritoneum and omentum has usually occurred, and the chances for survival are decreased. Other symptoms, such as leukorrhea, vaginal bleeding, or pressure signs, are usually disregarded by the patient. Frequent routine pelvic examination seems to offer the only means of early diagnosis. With surgical removal of the uterus, both tubes and ovaries, and the omentum, followed by intense roentgen irradiation of the pelvis, the five-year survival in this group was 20.28%. Eliminating cases in which complete surgical removal of the gross disease was impossible, the survival rate is about 50%.

Steroids in Cancer of the Breast.—Because of the effect of estrogens on breast cancer, Rosch and Green advocated castration of young women with breast cancer and have carried out this procedure in every possible instance. Castration, by x-ray or surgery, has produced a favorable effect on osseous metastases. After castration had its effect the authors resorted to the use of steroid hormones, chiefly androgens. Of the 50 women treated with androgens, three were also treated with estrogens. More than half of the patients showed subjective improvement, whereas only about a fourth showed objective improvement. The majority were given 100 mg. of testosterone propionate three times a week. One of three who were given only 25 mg. three times a week showed acceleration of metastases, which was controlled with 100 mg. three times a week. It was difficult to assess the relief obtained. In several instances, however, completely bedridden patients have become symptom-free and ambulatory. The authors combined steroids with radiation therapy, particularly in the presence of metastases in the long bones or weight-bearing areas. This study has not been in progress long enough to determine if there has been any change in longevity, although 20 of the patients were still alive at the time of writing. The majority who died did not show a favorable response to the initial course of steroid therapy. Steroids seemed to increase the length of life in those persons who demonstrated a definite objective response to combined steroid and x-ray therapy. In no instance was the disease cured by the administration of steroids alone.

Rocky Mountain Medical Journal, Denver

49:1-90 (Jan.) 1952

YOU AND YOUR A. M. A.—SYMPOSIUM

The A. M. A. Begins at Home. G. A. Unfug.—p. 24.
We Know Your Congressman. P. E. Wilson.—p. 26.
Things Are Happening in Rural Health. F. A. Humphrey.—p. 28.
You Are the A. M. A. E. J. McCormick.—p. 30.
Medical Education, Specialty Practice and Family Doctor. W. Darley. —p. 35.
Thorn Test During Contisone Therapy. G. C. Krebs and J. H. Holmes. —p. 40.
Cerebral Contusion of Newborn. R. M. Stuck.—p. 44.

Surgery, Gynecology and Obstetrics, Chicago

94:1-128 (Jan.) 1952

Combined Neck and Jaw Resection for Intraoral Carcinoma. W. W. Carroll.—p. 1.
Histopathology and Cytology of Cervix in Pregnancy. R. E. L. Nesbitt Jr. and L. M. Hellman.—p. 10.
Blood Volume in Pre-Eclampsia as Determined with P^{32} Labelled Red Blood Cells. N. I. Berlin, G. M. Hyde, J. H. Lawrence and others. —p. 21.
Effect of Stimulation of Lower Intrathoracic Portion of Vagus Nerve on Cardiovascular System of Dogs. S. T. Chester, H. C. Naffziger, C. Fisher and others.—p. 23.
Pancreaticoduodenectomy with Resection of Portal Vein in Macaca Mulatta Monkey and in Man. C. G. Child III, G. R. Holswade, R. D. McClure Jr. and others.—p. 31.
Results of Radical Surgical Extirpation in Treatment of Carcinoma of Esophagus and Cardia: With Five Year Survival Statistics. R. H. Sweet.—p. 46.
Transduodenal Reconstruction of Bile Ducts. L. R. Dragstedt and E. R. Woodward.—p. 53.
Asphyxia by Tracheobronchial Secretions. N. Ende and J. Ziskind.—p. 57.
Myolymphangioplasty for Prevention and Relief of Swollen Arm: Preliminary Report. N. Treves.—p. 65.
*Cytologic Interpretation of Prostatic Smear. H. Peters and I. N. Frank. —p. 69.
Problem of Calyceal Diverticulum. H. R. Newman, R. M. Lowman and L. L. Waters.—p. 77.
*Aortography: Its Interpretation and Value; Report of 200 Cases. R. Leriche, P. Beaconsfield and C. Boëly.—p. 83.
Roentgenology and Pathology of Cyclic Disturbances in Breast: With Special Reference to Their Surgical Diagnosis. J. Gershon-Cohen and H. Ingleby.—p. 91.
Hyperthyroidism in Pregnancy. M. E. Dailey and R. C. Benson.—p. 103.
Analysis of 100 Cases of Squamous Cell Carcinoma of Esophagus: Part II. With Special Reference to Its Theoretical Curability. K. A. Merendino and V. H. Mark.—p. 110.

Cytological Interpretation of Prostatic Smear.—This paper is concerned with the cytological interpretation of prostatic secretions obtained from 616 patients with various urologic disorders, including 100 patients who had carcinoma of the prostate. In 46 of the 100 patients the carcinoma was confirmed by histological examination of sections and in the other 54 patients cancer was indicated by clinical observations, such as roentgenologic evidence of metastases or elevated phosphatase values. Malignant cells were present in the prostatic smear of 88 of the 100 patients with carcinoma. Two of the 12 patients whose prostatic secretion smears failed to show malignant cells had been treated successfully with estrogen for several years. Malignant cells were reported to be present in two patients who on histological section proved to have benign prostatic hypertrophy. The finding of cancer cells in the smears of five patients led to the correct diagnosis earlier than would have otherwise been possible. In these cases the cytological investigation was of immeasurable help to the clinician and patient. The results of this survey show that cancer of the prostate desquamates cells sufficiently characteristic to be recognized microscopically. However, failure to find cancer cells in the smear does not exclude the presence of a malignant tumor. Adequate prostatic massage is essential for this study. A very hard prostate may fail to yield secretion on massage, and in other cases the gland may prove too tender to make adequate massage tolerable. If these mechanical obstacles are taken into consideration, the "false negative" report will become rare. The reliability of the cytological interpretation increases with the experience of the interpreter.

Evaluation of Aortography.—Many consider aortography a useful and safe method of investigation in selected cases which present diagnostic and therapeutic problems; some—fearing the needle puncture of the aorta—are unwilling to adopt this approach. Those who criticize aortography claim that the information obtained does not warrant the risks involved in this procedure. Of 2,000 patients with peripheral vascular disease observed by Leriche and associates during the last two years 190 were subjected to aortography. In addition to these, they performed 10 aortographies for conditions other than peripheral vascular disease. They feel that aortography is a procedure that may be carried out without risk to the patient, but its practical applications and values are limited. It is useful in a small group of patients with suspected abdominal or retroperitoneal tumors, in whom diagnosis is impossible to make by other simpler methods of investigation. In this group of patients with relatively normal arterial walls, aortography is done by the retrograde catheter method. In peripheral vascular disease, aortography should only be employed if resection or reconstructive surgery of the vascular lesion is to be considered. Here, the translumbar puncture of the aorta is the method of choice.

Urologic & Cutaneous Review, West Palm Beach, Fla.

55:715-780 (Dec.) 1951

*Genito-Urinary Tuberculosis. H. L. Kretschmer.—p. 715.
Nephrolithotomy for Staghorn Calculi. E. A. Brady Jr.—p. 724.
Myomatous Tumors of Kidney. S. Petkovic.—p. 730.
Patent Urachus. F. A. Lloyd and D. Pranke.—p. 734.
Some Newer Aspects of Analysis of Semen (Insemination Implication). R. D. Herrold.—p. 738.
Giant Hydronephrosis on Left Side of Horseshoe Kidney. H. J. Meisel and J. M. Miller.—p. 739.
Liposarcoma of Kidney. L. Brady and G. Wayburn.—p. 741.
Cavernous Hemangioma of Scrotum: Report of Case. W. Baurys and W. A. Morton Jr.—p. 744.

Genitourinary Tuberculosis.—Renal tuberculosis may produce no symptoms until the lesion is far advanced. Bladder symptoms predominate in most cases. Cystitis that does not respond to treatment, especially in young persons, should suggest the possibility of urinary tuberculosis. The diagnosis is based on demonstration of pus and tubercle bacilli in urine obtained by catheterization. Renal tuberculosis as seen by the urologist never heals, and necessitates nephrectomy. Improvement produced by streptomycin may be due to its effect on secondary bacterial invaders and not on the tuberculous process itself. Nephrectomy removes a large focus of tuberculosis, hence increases resistance and may delay the progress in the opposite kidney. Views on nephrectomy in bilateral renal tuberculosis have changed. The author generally refrains from nephrectomy in these cases, but when one of the two kidneys becomes acutely infected and the patient shows symptoms of sepsis, i. e., chills, fever, and sweats, nephrectomy is indicated. Tuberculosis of the bladder is never primary and must be considered with that of the kidney. The onset of genital tuberculosis is slow and insidious. Enlargement of the epididymis prompts the patient to consult his physician. A history of a slow painless growth, the presence of nodules in the epididymis, thickening and beading of the vas deferens, nodules and thickening of the prostate and seminal vesicles, and a previous abscess with the presence of a fistula or scar in the scrotum establish the diagnosis. The presence of a large amount of hydrocele fluid or extensive involvement of the epididymis with adhesions may cause difficulty in determining the condition of the testicle, but involvement of the testes is rare, and occurs late in the course of the disease. The patient should be operated upon as soon as the diagnosis of genital tuberculosis has been established, and as much tuberculous tissue as possible should be removed. The author sees no reason for a long course of preparatory treatment, pointing out that with local anesthetics or thiopental (pentothal®) sodium, the patient can be operated upon safely, even in the presence of active pulmonary tuberculosis. The conservative operation carried out most frequently is epididymovasectomy. As much of the vas deferens as possible should be removed, and the end of the vas should be cauterized with pure phenol. The radical operation consists of removal of the seminal vesicles, part of the prostate, the vasa, and both epididymides. Many urologists feel, however, that the excellent results obtained by conservative surgical methods do not justify the radical procedure.

FOREIGN

Archiv für Kinderheilkunde, Stuttgart

143:113-160 (No. 3) 1951

*Congenital Malaria. E. A. Voss.—p. 113.
Clinical Aspects of Dextroposition of Aorta During Childhood. H. Weyers.—p. 120.
Early Diagnosis of "Isolated" Tricuspid Atresia. G. Schaper.—p. 133.
Therapeutic Use of Pantothenic Acid in Children. W. Ehrengut.—p. 140.

Congenital Malaria.—Congenital malaria can be proved only when a mother who has malaria gives birth to a malarial child in a region where there are no Anopheles mosquitoes. Voss presents two cases of malaria that he believes can be classified as congenital and cites reports from the literature. Intrauterine infection of the fetus has been demonstrated repeatedly, but its mechanism has not been explained. The intact placenta acts as a filter, and plasmodial parasites that circulate in the blood cannot enter the tissues. It has been suggested that traumatic lesions of the placenta might be produced by malarial chills and other causes. The hemorrhagic tendency of malaria patients supports this assumption. The author believes that nonviability usually results in cases of intrauterine infection, and that, in infants born alive, infection generally takes place during delivery. Symptoms of malaria usually appear after a latent period of not less than seven days. Congenital malaria behaves like an inoculation malaria in that a single course of quinacrine is adequate for cure, supplementary treatment with pamaquine being unnecessary.

British Journal of Venereal Diseases, London

27:173-220 (Dec.) 1951. **Partial Index**

Serological Diagnosis of Lymphogranuloma Venereum. A. D. Macrae.—p. 183.
Serological Investigation for Treponemal Infection of Artisan Recruits in South Persia. R. Hodgson and I. S. Stewart.—p. 188.
Parallel Serum Testing with V. D. R. L. Slide Test, Meinicke Slide Test, and Price Precipitation Reaction. B. Singh and M. D. Sharma.—p. 190.
Parallel Testing with Crude Heart Extract Wassermann Antigens. I. N. O. Price and A. E. Wilkinson.—p. 191.
Case for Diagnosis with Note on Infantile Cortical Hyperostosis (Caffey's Syndrome). E. M. C. Dunlop.—p. 194.
Note on Use of Stuart's Transport Medium for Isolation of Gonococcus. A. E. Wilkinson.—p. 200.

British Medical Journal, London

1:1-58 (Jan. 5) 1952

Tissue Reactions to Protein Sensitization. J. R. Squire.—p. 1.
*Dental Disease Among 14-Year-Old London School-Children in 1947 and 1950. M. Mellanby and H. Mellanby.—p. 7.
Intestinal Macrocytic Anaemia. G. M. Watson and L. J. Witts.—p. 13.
Effect on Bleeding-Time of Extract of Blood of Patients with Rheumatoid Arthritis. R. Greene, J. Vaughan-Morgan and J. Gammon.—p. 17.
*Sterilization of Woolen Fabrics. H. F. Barnard.—p. 21.
Bacteriology of Vaginal Flora After Use of Internal Tampons. E. N. Brand.—p. 24.
T-Piece Technique for General Inhalational Anaesthesia: Safe Method of Reducing Operative Bleeding. J. Bullough.—p. 28.

Dental Disease Among 14-Year-Old Children.—Two surveys of 14-year-old London school children are reported. The inspections were made with a probe and mirror. The proportion of children found to be free from caries, according to the standards used, was 11.5% in 1947 and 16.1% in 1950. A total of 14.6% of the teeth in 1947 were carious, whereas for 1950 the figure was 13.9%. Only small differences were found between the two surveys as regards the amount of caries in the different types of teeth. The amount of treatment (fillings and extractions) that the teeth had received is briefly considered. An over-all reduction of 5% was noted in 1950 as compared with 1947. Some degree of gingivitis was found to occur in 73.7% of the children examined in 1947 and in 95.1% of those seen in 1950. Tartar deposits were frequently found, and there appeared to be some relation between the amount of tartar and the incidence and extent of gingivitis.

Sterilization of Woolen Fabrics.—For some years efforts have been made to reduce cross infections in hospitals, most of which are caused by Staphylococcus aureus. Periodical checking of the staff has shown that about 50% are nasal carriers of this organism. However, phage-typing of the organisms recovered from patients and staff cast doubt on the direct nurse-to-patient route of spread. Therefore, when the majority of freshly laundered blankets were found to contain Staph. aureus, methods of chemical sterilization were investigated. Effective concentrations of the detergent cetyl pyridinum bromide (also known as "fixanol C") were determined. The detergent on the dried blanket readily passes again into solution on contact with moisture. With a 1 in 6,400 dry concentration, the blanket leaves the laundry virtually sterile. Moreover, it apparently retains for a considerable period sufficient material to destroy any staphylococci that may be picked up in secretions. The impregnated blankets have a slightly smooth feel and no smell, and in routine hospital use of all blankets for a year there has been no indication of sensitivity of patients to the treated blankets. However, after the hospital blankets had been sterilized for three months, a certain amount of cross infection still continued, apparently from other sources.

Canadian Medical Association Journal, Montreal

66:1-94 (Jan.) 1952

Anxiety: Damage and Repair. D. E. Cameron.—p. 1.
Value of Aureomycin in Prevention of Cross Infection in Hospital for Sick Children. C. E. Snelling and R. Johnson.—p. 6.
Treatment of Dehydration in Infancy Using Continuous Interstitials and Hyaluronidase. A. M. Goodfellow, R. Pocock and J. F. McCreary.—p. 8.
Thiamin Therapy in Neuro-Psychiatry. I. Gregory.—p. 12.
Chloromycetin in Treatment of Mumps. G. Nickerson and E. M. Worden.—p. 17.
Our Present Knowledge of Potassium in Physiological and Pathological Processes. J. W. Stephens.—p. 19.
Spinal Analgesia with Hypobaric Pontocaine for Caesarean Section. R. A. Chaplin.—p. 32.
Unilateral Renal Agenesia. W. P. Hogarth.—p. 34.
Pain About Radial Styloid. A. C. Walsh and G. E. Miller.—p. 36.
Keratosis of Larynx with Progression to Malignancy. A. A. Grossman and W. H. Mathews.—p. 39.
Invasive Hydatidiform Mole. A. Gaum.—p. 42.
Observations on Oral Administration of Cortisone Acetate. M. A. Ogryzlo and A. G. Gornall.—p. 44.
Prevention in Control of Glaucoma. H. Pichette and J. Audet.—p. 48.
Eosinophil Level in Psychiatric Conditions. A. Mann and H. Lehmann.—p. 52.
Primary Amyloidosis Simulating Cirrhosis of Liver. W. Graham.—p. 58.
ACTH and Cortisone Therapy in Eye Disease. H. L. Ormsby, S. Y. Shirley, J. F. Aikenhead and R. G. Murray.—p. 62.

Klinische Wochenschrift, Heidelberg

30:1-48 (Jan. 1) 1952. **Partial Index**

*Are Nicotinic Acid Preparations Effective in Treatment of Peripheral Circulatory Disturbances? W. Schulze.—p. 8.
Oxidation of Anomeric Sugars. G. Malyoth and H. W. Stein.—p. 14.
*Cyclical Changes in Sizes of Nuclei in Vaginal Smears. H. Malsch and A. Schmitt.—p. 15.
*Counting Thrombocytes Under Fluorescent Light. W. Kosenow.—p. 18.
Electrical Charge and Isoelectric Point of Blood Cells: Relation to Cellular Function. E. Fritze and P. Doering.—p. 19.
Problems of Determination of Prothrombin. P. Arnolds and E. Erlemann.—p. 22.
Modification of Blood Coagulation by Streptomycin, *p*-Aminosalicylic Acid and Thiosemicarbazone. G. Walther and K. A. Winter.—p. 25.
Radioactive Phosphorus in Therapy on Polycythemia Vera. H. Goldeck, H. Groth and W. Horst.—p. 28.
Pathogenesis of Hyperglobinemic Purpura. F. E. Schmengler and H. Esser.—p. 30.

Nicotinic Acid and Circulation in Extremities.—Schulze believes that nicotinic acid stimulates the peripheral circulation only in the head and the upper part of the body including the proximal portion of the extremities. However, some clinicians think that circulation in the ends of the extremities is also stimulated by nicotinic acid, and that therefore this acid and its derivatives may be used in treatment of impaired circulation in the fingers or toes. Schulze measured the skin temperature of fingers and toes under conditions that would disclose vasoconstriction as well as vasodilatation. His experiments revealed that, whether nicotinic acid or its derivatives were given intravenously, intramuscularly or orally, they did not increase the blood perfusion of fingers or toes. On the contrary, the finger temperature often decreased in healthy subjects by as much as 10 C and more.

Observations on patients with disturbances in the peripheral circulation also proved that nicotinic acid preparations are not suitable for the treatment of impaired circulation in fingers and toes.

Cyclical Changes in Sizes of Nuclei in Vaginal Smear.—In 13 women who had fairly regular menstrual cycles, vaginal smears were made every second day throughout a whole cycle. The smears were fixed and stained according to the technique of Papanicolaou. In every smear at least 200 nuclei were measured and grouped into six classes according to the size of the nuclei. Although these classes of nuclei do not correspond completely to the five types of cells that Papanicolaou has ascribed to the various epithelial layers of the vagina, the nuclei of classes 5 and 6 correspond to the epithelial cells of the surface zone and the nuclei of class 1 to the epithelial cells of the middle and lower layers. On the basis of the studies on 13 women, in whom a total of about 30,000 nuclei were measured, the authors conclude that the epithelial cells with the smallest nuclei increase in number around the middle or at the beginning of the second half of the cycle. There is a second peak after the menstrual flow ceases. The number of macrocellular epithelial cells vary inversely as the number of parvicellular epithelial cells. These observations clearly indicate cyclical changes in the sizes of the nuclei in the vaginal smears.

Counting of Thrombocytes in Fluorescent Light.—The most widely used method at present for counting thrombocytes is that of Fonio, which has proved adequate for practical diagnosis. Kosenow's aim is not to replace this technique by a new one, but only to make it more rapid and easy with the aid of the fluorescent microscope. Auramin and thioflavin S were found to be the most satisfactory dyes. Inspection is done under oil immersion with an ordinary microscope, but the room should be dark if possible. For illumination the author employs a carbon arc lamp and a fluorescent lamp that has a high pressure mercury burner. Special filters are also desirable. Numerous comparative tests revealed that thrombocyte counts in specimens stained with fluorescent dyes were essentially the same or slightly higher than those by the Fonio method, because in the fluorescent light thrombocytes are less likely to be overlooked. The author stresses that with this method staining is rapid and simple, there is good contrast in the picture, and counting is rapid.

Lancet, London

1:1-60 (Jan. 5) 1952

Approach to Specialism. E. Finch.—p. 1.
Mediastinitis Following Foreign-Body Perforation of Cervical Oesophagus. F. B. Korkis.—p. 4.
*Comparison of Oral and Subcutaneous Administration of Methonium Salts in Treatment of High Blood-Pressure. J. A. Kilpatrick and F. H. Smirk.—p. 8.
*Green Sputum. A. J. Robertson.—p. 12.
Hysterosalpingography in Female Infertility: Comparison of Lipiodol and Viskiosol Six. D. Freeth.—p. 15.
Trial of Pituitary Growth Hormone in Case of Hyperinsulinism Due to Islet-Cell Adenoma. K. O. Black, I. Macdougall, E. Reid and F. G. Young.—p. 19.
Indications for Barium Enema in Diagnosis of Carcinoma of Colon. M. Haggie.—p. 21.
Isolation and Properties of Third Clotting Factor in Blood-Platelets. S. van Creveld and M. M. P. Paulssen.—p. 23.
Colonic Replacement of Stomach: Early Results of Radiological Investigation. B. B. Harrison.—p. 25.
Underfeeding as Factor in Infantile Vomiting. B. S. B. Wood.—p. 28.

Oral and Subcutaneous Administration of Methonium Salts.—The general impression from the literature is that, despite the great convenience of oral medication, fewer patients fail to respond to subcutaneous administration of hexamethonium. Kilpatrick and Smirk describe the results obtained in the same group of patients with subcutaneous and oral administration of hexamethonium bromide. They found that much larger doses of hexamethonium bromide are needed by the oral than by the subcutaneous route. The effects of oral administration continue longer, but are less predictable. Symptoms of intolerance, such as malaise, diarrhea, abdominal distension, nausea, vomiting, and occasionally collapse, are observed in a large percentage of the orally treated patients. Oral therapy is more likely to be successful when the subcutaneous dose needed to control blood pressure is small, either from natural sensitivity, after sympathectomy, or where excretion is delayed, as in some patients with chronic nephritis. Sometimes a salt-poor diet makes oral therapy possible with hexamethonium bitrate. Patients whose blood pressure reduction is inadequate or who have important side effects from hexamethonium bromide orally should be maintained on the insulin-like regime with hexamethonium bromide subcutaneously. The best results can be obtained only by adjusting the dose, times of administration, and mode or modes of administration in terms of day-long observations of the patient's response. Failure to decrease hypertension with hexamethonium salts is usually due to a defect in technique, such as neglect to make use of the more satisfactory reduction in blood pressure in the standing and sitting postures, neglect of the changing sensitivity to hexamethonium bromide, incorrect usage, or unwillingness to use the subcutaneous route when the oral route is unsatisfactory.

Green Sputum.—Robertson tried to find out why sputum became green and the relationship of such sputum to bronchiectasis by collecting some 50 samples of green sputum from patients with bronchiectasis or pulmonary abscess. After much trial and error the method adopted was to centrifuge the sputum, preferably after allowing it to stand for some time, and then to emulsify the greenish upper layer with chloroform, and again centrifuge for a long time. In this way it was possible to get a green solution that was clear enough for spectrophotometry. The absorption curves in the spectrum were those of verdoperoxidase, and the author suggests that green sputum is purulent sputum in which there is failure to excrete verdoperoxidase, which accumulates and colors the sputum green. This happens whenever there is stagnation, especially in disease of the bronchial wall. Two clinical examples may be given: (1) during the night the patient may not cough much, and by morning his sputum may be green, but once this has been expectorated the purulent material may revert to its normal color unless there is continued difficulty with expectoration; and (2) in a lung abscess there is destruction of leukocytes with failure to excrete any enzyme, and a green color may be found when the abscess is coughed up.

Medical Journal of Australia, Sydney

2:761-796 (Dec. 8) 1951. Partial Index

Rheumatoid (Ankylosing) Spondylitis: Its Early Manifestations and Early Diagnosis. J. H. Young.—p. 761.
Congenital Obstruction of Nasolachrymal Duct: Review of 130 Cases. J. B. G. Gibson.—p. 764.
Cross-Infection of Surgical Wounds. P. M. Rountree.—p. 766.
Clinical Observations on Series of Cases of Advanced Malignant Disease Treated with Antibiotics and ACTH. F. A. Maguire and M. McElhone.—p. 769.

2:797-832 (Dec. 15) 1951

Examples of Use of t-Test on Physiological Data. C. White.—p. 797.
Appendicitis in Childhood. E. S. Stuckey.—p. 804.
*Observations on Ethylidene Dicoumarin as Anticoagulant, with Special Reference to Puerperal Patients. W. McI. Rose and V. I. Krieger.—p. 813.

Ethylidene Bishydroxycoumarin in Puerperal Patients.—The ethylidene homologue of bishydroxycoumarin, which is 3,3′-ethylidene-bis (4 hydroxycoumarin), has the following advantages over bishydroxycoumarin: (1) it is less toxic than dicoumarin: (2) the recovery from hypoprothrombinemia is more rapid, being almost complete within 24 hours instead of three or more days; and (3) hypoprothrombinemia can be prolonged by continuous administration. This paper reports on the prophylactic and therapeutic use of this drug. The 400 patients who were given ethylidene as a prophylactic measure included 364 women who underwent cesarean section and 36 who had a hysterectomy. The group of patients in whom the drug was used for treatment included 16 with thrombosis of the femoral artery, 14 with calf vein thrombosis, 12 with pulmonary infarction, and 11 with cardiac infarction. The drug is administered by mouth in the form of 100 mg. tablets. Treatment is usually begun with a dose of 500 mg. on each of the first two days and 300 mg. on the third day. Further dosage is regulated by the prothrombin level of the blood, which is determined three

times a week. At first an attempt was made to keep the prothrombin level below 30% of the average normal level, but 50% was adopted in later cases. From the results obtained the authors conclude that ethylidene bishydroxycoumarin is as safe and yet as effective as bishydroxycoumarin. The clinical results also show that ethylidene bishydroxycoumarin may be used in the last eight weeks of pregnancy without ill effect to either mother or child.

Medicina, Mexico

31:471-494 (Dec. 10) 1951. **Partial Index**

Treatment of Rheumatoid Arthritis with 11-17 Oxysteroids, Synergistic Substances, and Substitute Substances. J. Torres Morales.—p. 471.
*Results of Penicillin Treatment of Mal del Pinto After Two Years. C. Rein, D. Kitchen, F. Marquez and G. Varela.—p. 488.

Results of Penicillin Treatment of Pinta After Two Years.—Microcrystalline procaine penicillin G in oil with aluminum monostearate was used in treating 665 patients with pinta. The Treponema carateum disappeared from the skin in about six hours following injection of from 300,000 to 1,200,000 units. Much longer periods were required for the disappearance of the parasites when aureomycin, dihydrostreptomycin, chloramphenicol, and terramycin were used. Clinical and serological examinations of 350 of the patients were carried out at intervals of from one to three months for two years. Serological response takes place much more slowly in pinta than in syphilis or yaws, perhaps because the disease has usually been present for a long time before treatment is sought. Clinical results are also less rapid, five or six months being required for the disappearance of the pintids. The results obtained with one injection of 4 cc. (1,200,000 units) are better than those obtained with one injection of 1 cc. weekly for four weeks, although the total dosage is identical. At the end of the two year period, 20% of the patients had a negative serological response; 55% showed marked improvement; and in 25% the serological reaction was unchanged.

Nordisk Medicin, Stockholm

46:1779-1810 (Nov. 28) 1951. **Partial Index**

*Diagnosis and Treatment of Tumors of Bladder. G. Giertz.—p. 1779.
Intravenous Nutrition with Amino Acids. F. Lidström and A. Wretlind. —p. 1783.
Fixation Treatment of Fractures of Carpal Scaphoid Bone. S. Wallensten. —p. 1789.
Lymphogranulomatosis of Heart. Case of Hodgkin's Sarcoma of Heart. O. A. Sydnes.—p. 1794.
Torsion of Gallbladder. S. E. Ekdahl.—p. 1796.

Diagnosis and Treatment of Bladder Tumors.—Giertz treated 409 fibroepitheliomas of the bladder from 1940 to 1948. The tumors varied from benign papillomas to highly malignant solid tumors. In so far as possible the degree of malignancy, the size, depth of growth, and possible presence of metastases must be determined in judging a tumor of the bladder. The prognosis is believed to depend chiefly on the incidence of metastases. The deeper the tumor penetrates, the graver the prognosis. Necropsy in 67 of the cases (Franksson) revealed no metastases when the tumor was limited to the submucosa and metastases in only two out of eight cases in which the tumor had grown into the musculature but had not penetrated the wall, but there were metastases in more than four-fifths of the cases with deep perivascular growth. Of the patients with tumors limited to the mucosa, 83%, of those with tumors limited to the submucosa, 70%, and of those with tumors penetrating the musculature, only 20% were living after five years. In patients with tumors of the perivascular growth only 1 of 17 lived three years, and none lived five years. The author's impression is that the tumors retain their original character and degree of malignancy. Radiotherapy has but slight effect. Benign tumors limited to the mucosa are treated radically by removal of the tumor and the underlying mucous membrane by transurethral or transvesical intervention. With larger benign tumors suprapubic operation is performed to a large extent, but in recent years the transurethral method has been applied. With malignant tumors still limited to submucosa, cystectomy is not generally necessary. If the tumor has grown into but not through the musculature, resection is preferred whenever it is technically possible to remove the tumor by partial extirpation of the bladder. Since often it cannot be determined beforehand whether or not resection is possible, all patients are now prepared for ureter implantation, to allow the choice at operation between resection and total cystectomy. If the tumor has invaded the perivascular tissue it is questionable whether operation should be attempted, but total cystectomy is carried out if the tumor is not too extensive and fixed and if the general condition is good. Continued systematic research is necessary to establish what forms of treatment under different circumstances give the best results.

Presse Médicale, Paris

59:1697-1716 (Dec. 22) 1951

400 Cases of Allergic Bronchial Asthma; Statistical Study. Pasteur Vallery-Radot and P. Blamoutier.—p. 1697.
*Pain in Thoracic Portion of Vertebral Column Associated with Gynecologic Syndromes Characterized by Excessive Quantities of Estrogens. A. J. Bret and M. Bardiaux.—p. 1698.
Various Stages of Biological Diagnosis of Virus Pneumonia. H. Boucher, J. Bertein and J. Vailhé.—p. 1700.

Dorsalgia in Gynecologic Syndromes.—The occurrence of spontaneous pain, in general localized between the scapulas in the thoracic portion of the vertebral column and frequently radiating unilaterally to the intercostal spaces, has been reported in 24 women. Thirteen had never had children, while the remaining 11 had 18 children, 1 extrauterine pregnancy, and 13 abortions. They were from 14 to 58 years old, but as a rule the first attack of pain occurred between the ages of 25 and 40. Sometimes it was dull and hardly perceptible and sometimes it was lancinating and paroxysmal. Occasionally the pain was elicited by leaning against the back of a chair, or it was preceded by work with the upper extremities or by sudden and prolonged strain. The thoracic pain was frequently associated with pain in the lumbosacral region. Roentgenologic examination of the vertebral column frequently revealed that lesions had been present from adolescence or were congenital, such as complete sacralization of the fifth lumbar vertebra or hemisacralization. Flattening of the thoracic vertebrae with a squamous aspect of the cartilage was observed in 10 patients. The dorsalgia was associated with functional disturbances of the sex organs, such as painful breasts, menstrual disturbances, or vaginal discharge. Biopsies of endometrium were made in nine patients, and revealed absence of luteinization in five. The temperature of three patients was elevated, with 99 F during the first part of the cycle in one, and with 100.4 F to 101.2 F during the first two days of menstruation in two. Of 22 patients who had sexual relations, 21 complained of frigidity following the onset of their disturbances. Treatment with estrogens seemed to have elicited or aggravated the disturbances in 9 of the 24 patients. Pain subsided rapidly after injections of progesterone and oral administration of tablets of methyl testosterone, but frequently the favorable results were only temporary.

Revue de la Tuberculose, Paris

15:917-1064 (No. 10-11) 1951. **Partial Index**

Critical Study of Concept of Primary Complex in Primary Tuberculous Infection. R. Kourilsky, M. Bidermann and S. Ettedgui.—p. 917.
Comparative Results of Culture and Smear Examination for Detection of Koch's Bacilli. A. Meyer, R. Galland and B. Kobrinsky.—p. 932.
*Synergistic in Vitro Effect of Streptomycin, *p*-Aminosalicylic Acid, Viomycin and Neomycin in 11 Combinations on Mycobacterium Tuberculosis. Coletsos.—p. 957.

In Vitro Synergism of Antibiotics on Mycobacterium Tuberculosis.—Four strains of Mycobacterium tuberculosis, two of which were resistant to streptomycin, were tested in vitro in a Löwenstein-Jensen medium to which streptomycin, sodium *p*-aminosalicylate, viomycin, and neomycin had been added in 11 different combinations. Results showed that in general, there was intensified synergism when two antibiotics were employed, simple synergism when three were employed, and no evident synergism when all four were used. Best antibiotic combinations

were in the order as follows: streptomycin combined with sodium *p*-aminosalicylate first, streptomycin combined with viomycin second, and sodium *p*-aminosalicylate combined with viomycin last. A combination of all three antibiotics should be avoided.

Riv. di Pat. e Clin. della Tuberc., Bologna

24:257-320 (Sept.-Oct.) 1951

*Evaluation of Anatomic and Functional Condition of Heart in Pulmonary Tuberculosis. L. Costantini.—p. 257.

Test of Constellation of Wuhrmann, Wunderly, and De Nicola (Cadmium Sulfate Reaction-Weltmann's Reaction) on Globulin Fractions of Serum, and Erythrocyte Sedimentation Reaction in 133 Cases of Pulmonary Tuberculosis. F. Nobile.—p. 265.

*Mediastinal Pleurisy. B. Cossar and F. Antonini.—p. 286.

Histamine Factor of Tuberculin Reaction: Critical Review. F. Malaguzzi Valeri.—p. 296.

Cardiac Function in Patients with Pulmonary Tuberculosis.—Cardiac function was studied in 40 patients with pulmonary tuberculosis by means of electrocardiograms taken before and after administration of ouabain. In 10 patients with exudative tuberculosis no heart lesions were known to be present; in 10 with cavitary tuberculosis and widespread bilateral involvement there were vague clinical signs of slight cardiovascular insufficiency; in 20 with ulcerative tuberculosis, of whom 10 were undergoing pneumothorax, signs of cardiac insufficiency had been demonstrated by prior electrocardiograms. Tachycardia was present in almost all the patients. In the morning one electrocardiogram was obtained with standard leads from the fasting patient. Additional electrocardiograms were taken 5, 15, and 30 minutes after intravenous injection of 0.25 mg. of ouabain. The condition of the heart was in general good in patients with exudative tuberculosis. Myocardial lesions were found most frequently in the 10 patients with cavitary tuberculosis. These were revealed by a deranged auriculoventricular conduction, an altered spreading of the intraventricular excitation, and definite pathological repolarization attributable to a coronary insufficiency that was accentuated by the ouabain. The tracings were less consistent and less definite in patients with ulcerative tuberculosis, but they revealed that the disease impairs the function of the left ventricle. The bradycardic action of ouabain was observed in one patient with exudative tuberculosis and in four patients with ulcerative tuberculosis. Tachycardia was observed in four patients with cavitary tuberculosis and in two patients with ulcerative tuberculosis. The myocardial lesions may be due to the tuberculous infection of the heart muscle and to a diminished supply of oxygen to the myocardium. The disturbances of rhythm are possibly due to a reaction of the vagus nerve to stimulation of the cerebral centers impaired by anoxia. These and other causes may have a pronounced harmful action on the heart that, however, cannot be classified as cor pulmonale because it would not be justified by the clinical and electrocardiographic manifestations. A definite diagnosis and prognosis of the condition of the heart in patients with pulmonary tuberculosis is difficult, and all available means should be used in the attempt to detect concealed heart lesions in these patients. Ouabain proved to be a good adjuvant in the diagnosis.

Mediastinal Pleurisy.—The authors review extensively the clinical, roentgenologic, and anatomicopathological aspects of pleurisy involving the mediastinum. Mediastinal pleurisy may be central, posterior, anterior, superior, inferior, right, and left according to the area of the mediastinum that is affected. It may be acute or chronic, with or without effusion. Exudative mediastinal pleurisy may be serofibrinous, purulent, and hemorrhagic. The causative agents include tubercle bacilli, Treponema pallidum, and pneumonococci. Clinically a definite diagnosis of mediastinal pleurisy cannot be established without a roentgenographic investigation of the chest. Subjective symptoms in severe cases may be retrosternal pains extending to the breast and the axillary area, while the commonest objective finding may be contralateral deviation of the trachea because of compression of the exudate against the mediastinal organs. The typical roentgenographic finding is a triangular shadow of increased density with varying positions of the margins according to the area of the mediastinum involved. In addition, the roentgenogram reveals collateral phenomena, such as displacement of the mediastinal organs and hypomotility of the diaphragm, that make diagnosis difficult. Differentiation of this disease from paravertebral osteolytic abscess, pericarditis, esophageal diverticulitis and megaesophagus, retrosternal goiter, hypertrophic thymus, lymphogranuloma and lymphatic leukemia localized in the mediastinum, and other diseases is difficult. Clinical and physical signs, bronchography and body section roentgenography, puncture of the pericardium, hematological findings, and biopsy help to determine the correct diagnosis. Rotation of the patient during roentgenologic investigation is indicated. The authors reported 19 instances of mediastinal pleurisy on 7,968 routine examinations of the chest (an incidence of about 0.24%) during the period of 1947-1950 in the hospital of Addis Ababa. In only one-third of these patients was a diagnosis of mediastinal pleurisy considered before roentgenograms were taken.

Thorax, London

6:343-454 (Dec.) 1951

Observations on Normal Anatomy of Bronchial Arteries. L. Cudkowicz and J. B. Armstrong.—p. 343.

Anatomy and Applied Anatomy of Mediastinal Fascia. P. Marchand.—p. 359.

Pneumonectomy with Immediate Thoracoplasty in Treatment of Pulmonary Tuberculosis. G. Cruickshank and E. Papamichael.—p. 369.

Pulmonary Resection for Tuberculosis in Children. C. A. Ross.—p. 375.

Rupture of Oesophagus. B. V. Slesser.—p. 389.

Surgical Treatment of Bronchial Fistulas After Lung Resection for Pulmonary Tuberculosis. O. Monod, G. Babou and J. Lo.—p. 397.

Thoracic Chordoma. G. G. Crowe and P. B. L. Muldoon.—p. 403.

Bronchography in Children. M. Bates, I. C. W. English, H. M. Foreman and T. M. Wilson.—p. 408.

Importance of Sleeping Posture in Spread of Pulmonary Tuberculosis. W. H. Helm.—p. 417.

Chronic Diffuse Interstitial Fibrosis of Lungs. A. G. Heppleston.—p. 426.

Study of Open Circuit Method of Intrapulmonary Mixture of Inspired Air. M. R. Becklake.—p. 433.

Ugeskrift for Laeger, Copenhagen

113:1653-1686 (Dec. 6) 1951. **Partial Index**

Investigations on Disinfection of Wounds. K. T. Kjølhede, J. Bock and E. Lund.—p. 1657.

*Psychoses Following Antabuse Treatment. P. J. Rasch.—p. 1658.

*Dosage of Antabuse® with Special Regard to Avoidance of Side-Effects. O. Martensen-Larsen.—p. 1660.

Psychoses Following Antabuse® Treatment.—The three cases of psychosis in chronic alcoholics are attributed to the tetraethylthiuramdisulfide (antabuse®) medication. The psychoses were usually marked by excited confusion, but at times also by apathy, which in two cases represented a terminal phase in the course of the psychosis and in one case increased to stupor. The recurrent signs of paresis seen in the two patients who passed through a definite apathetic or stuporous phase disappeared spontaneously after a longer time. In all cases there had apparently been an overdose of the substance because the subjective distaste for alcohol was not considered sufficient; later experience showed that the attained effect had been fully sufficient. Rasch urges careful dosage and attention to early psychic symptoms during antabuse treatment.

Dosage of Antabuse® with Special Regard to Avoidance of Side-Effects.—Martensen-Larsen warns against unnecessarily large doses of tetraethylthiuramdisulfide (antabuse®). On test with light beer (or corresponding amount of alcohol) there should be only a slight flush, feeling of warmth, and increase in pulse. The patients should consult the physician at stated intervals. Since the effect of the drug may increase during the first three months, depending on the regular intake, no final minimal maintenance dose must be accepted as satisfactory before this period is over. At every consultation attention must be directed to possible side-effects, which must be borne in mind in determining the continued dosage. Tetraethylthiuramdisulfide can be regarded as a useful and safe means of treatment only if these considerations are followed.

BOOK REVIEWS

Modern Practice in Infectious Fevers. Vols. I & II. Edited by H. Stanley Banks, M.A., M.D., F.R.C.P., Senior Physician, Park Hospital, Hither Green, London. Cloth. $20 per set. Pp. 496; 499-989, with 211 illustraticns. Paul B. Hoeber, Inc. (medical book department of Harper & Brothers), 49 E. 33rd St., New York 16, 1951.

This book, in general, offers a good discussion of acute infectious diseases. The individual chapters have been written by 52 contributors, chiefly from the British Commonwealth, with a few from America. The first part of the first volume is devoted to a general approach to infections, beginning with an interesting chapter from a general biologic point of view on the distribution in time and space of the various types of microorganisms. Short articles follow on laboratory aids to diagnosis and treatment. The value of the short section on biochemical methods is questionable in a book of this scope. Immunization against the various fevers is included in separate articles under a single chapter heading and is not discussed under the individual diseases. The remainder of volume 1 is taken up with the discussion of bacterial fevers. Special emphasis is placed on the bacteriology and pathology of the particular infections. The discussions of general measures, including supportive therapy, are not as detailed as the discussions of specific therapy. In spite of the inclusion of a chapter on chemical methods, the biochemical changes in specific diseases, such as the alterations in the blood chlorides in pneumonia, are largely omitted. Tuberculosis and the venereal diseases are excluded except for a discussion of tuberculous meningitis.

The second volume includes the discussions of viral, rickettsial, spirochetal, protozoal, and systemic mycotic diseases, as well as those diseases of unknown etiology that resemble infections, such as infectious mononucleosis. Most of the articles have short lists of references appended to them; in a few articles, the references are extensive. The illustrations are well chosen and well reproduced. The electron micrographs are particularly noteworthy, and the photomicrographs of pathological preparations are good. The clinical photographs often have distracting backgrounds, but the lesions are well shown. The design and reproduction of the graphs and charts depicting the clinical course of patients are usually poor. The color plates are uniformly good. The book should serve its purpose well as a guide to practitioners who may be treating acute infections.

Brain Metabolism and Cerebral Disorders. By Harold E. Himwich, M.D., Chief, Clinical Research Branch, Medical Division, Army Chemical Center, Maryland. Cloth. $6. Pp. 451, with 53 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1951.

The author of this book takes for granted that the brain is a chemical machine. To the uninitiated, it may come as a shock to learn that the brain is not as different from muscle as muscle is from the liver. In this era in which psychosomatic medicine receives so much emphasis, it is hard to believe that what we are, what we think, and what we do depends on a great deal of highly complicated chemistry. This view may seem unsatisfactory and unprofitable to some, but it is the correct view for the modern scientific physician, and it becomes increasingly profitable as new techniques are developed for observing the minute and rapid changes that occur in the chemistry of the cerebral neurons. For the first time, we are in possession of sufficiently refined tools to provide an inside view of brain function. The electron and the radioactive tracer are the functional probes of the neurochemist, the new "neuroanatomist," who carries the description of central nervous organization beyond the gross and static histological range to the ultramicroscopic and highly dynamic chemical range.

Dr. Himwich is in the forefront of the neurochemists; he has his eye on the future, but, in this book, he organizes the experience of the past. The scope of this book is vast, for it starts with a discussion of molecular structure and works its way up through phylogeny and ontogeny to the highest levels of nervous function. Electroencephalography is considered because the author accepts the electrical activity of the brain as a useful indicator of brain function. Autonomic, sensory, motor, and associative functions are considered because disorders of brain metabolism can manifest themselves in disturbances of function in these spheres. In the present state of our knowledge, so vast a work is necessarily full of gaps. It is really surprising that with so much crucial information lacking the author has been successful in weaving a general pattern on a coherent fabric of fact. The work is written in essay style and is surprisingly easy to read. Though a strictly scientific attitude is maintained throughout, the author's manner is engagingly informal.

The book is divided into two parts. The first is concerned with energetics and the second with patterns of nervous activity. The contents include the following topics: foodstuff of the brain, mechanisms for maintaining the carbohydrate supplies of the brain and for maintaining brain metabolism during hypoglycemia, the oxidation of carbohydrate in the brain, aspects of cerebral circulation, cerebral metabolism during growth of lower mammals, a biochemical basis for neurophylogenesis, human cerebral metabolic rate, therapeutics and its application to brain metabolism, the somatic division of the central nervous system, pattern of activity in the five phyletic layers, the barbiturates and other depressant drugs, a classification of clinical signs and a theory of narcosis, and prospects and problems for the future as well as a bibliography and an index.

Reading is facilitated by a summary at the end of each chapter. The diagrams, figures, and tables provide a systematic arrangement of important qualitative and quantitative data that prior to this publication were scattered widely through the medical and chemical literature and were, therefore, generally unavailable. One longs at times, toward the middle and end of this book, for luminous simplifications and wonders whether judicious omissions would not have been helpful; however, the work has an almost biologic complexity that is more appropriate than artificial simplification and schematization. Everything is included, good and bad, old and new material, speculation and fact. Some irreconcilable paradoxes are left hanging in the air to the admitted discomfort of the author. A bibliography of over 1,000 titles is skillfully woven into the text. This bibliography is unique and is worth the price of the book.

For the brain chemist, this is an invaluable reference work. For the anesthesiologist and the interested physician, it is a one-volume library of the best that has been thought and said on the subject of the metabolic basis of brain disorders.

Global Epidemiology: A Geography of Disease and Sanitation. Volume II: Africa and the Adjacent Islands. By James Stevens Simmons, B.S., M.D., Ph.D., Dean and Professor of Public Health, Harvard University School of Public Health, Cambridge, Mass., Tom F. Whayne, A.B., M.D., M.P.H., Colonel, M.C., United States Army, Gaylord W. Anderson, A. B., M.D., Dr.P.H., Mayo Professor and Director, School of Public Health, University of Minnesota, Minneapolis, Harold Maclachlan Horack, B.S., M.D., Instructor in Medicine, Tulane University School of Medicine, New Orleans. Associate author: Ruth Alida Thomas, A.B., M.A., M.P.H., Research Associate, School of Public Health, University of Minnesota, Minneapolis, and collaborators. Cloth. $15. Pp. 652, with illustrations. J. B. Lippincott Company, 227-231 S. Sixth St., Philadelphia 5; Aldine House, 10-13 Bedford St., London, W.C.2; 2083 Guy St., Montreal, 1951.

In this volume, Africa and the adjacent islands are discussed. Until World War II, this field was almost unexplored, as data concerning contagious and epidemic diseases as well as other important material from the far flung countries of the globe had never been brought together, so that it could be referred to readily. Although much of this material was available in the United States, it was scattered in all kinds of books, journals, and documents, so that it required a great deal of research to glean anything of value for even one country.

The medical department of the United States Army undertook to bring together this material as a project of the Division of Preventive Medicine in the Office of the Surgeon-General. The

The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

group working here, under the leadership of Brigadier General James S. Simmons, rapidly expanded, so that various fields of public health as applied to global epidemiology were studied.

In the discussion in this volume, Africa has been divided into eight sections and is considered under the influence of various factors, such as geography and climate, population and socioeconomic conditions, environment and sanitation, health services and medical facilities, and a consideration of the diseases that occur in the various sections. There is an excellent annotated bibliography at the end of each section. The one on the Belgian Congo, for instance, includes 69 references. The territory known as Ruanda-Urundi, a section that few Americans have ever heard of, is described in seven pages of text, with 22 bibliographic references.

This book would be of unestimable value if military operations were purposed in any part of Africa by our armed forces; however, it is not necessary to wait for military operations to prove its usefulness, as any person or firm contemplating visiting Africa for any length of time would do well to have this reference book, in order to determine such things as types of clothing best suited to the country, what insects one must protect against and what diseases might occur, if adequate precautions are not taken. It is a book that should be in all reference libraries.

Diseases of the Nervous System. By W. Russell Brain, D.M., F.R.C.P., Physician to London Hospital and to Maida Vale Hospital for Nervous Diseases, London. Fourth edition. Cloth. $8.50. Pp. 1002, with 85 illustrations. Oxford University Press, 114 Fifth Ave., New York 11; Amen House, Warwick Sq., London, E.C.4, 1951.

This edition has appeared four years after the last edition. The book has been popular with students and neurologists. This edition brings the book up-to-date, particularly with regard to therapy. There are new sections on leptospiral meningitis, temporal arteritis, infectious mononucleosis, and sarcoidosis. From whatever standpoint we look at this work, it is good. What has been said (*J. A. M. A.* **137**:1348 [Aug. 7] 1948) of the third edition can be repeated: ". . . the amount of information offered is enormous. It is good for the student, good for the practitioner, and good for the diehard neurologist." The editorial work deserves particular praise. The whole book contains only a few insignificant misspellings. Any criticism of the book can concern only minor items. For example, though the terms "symptoms" and "signs" are used, the author for the most part appears to forget that symptom is a subjective and sign an objective phenomenon. Wasting and fasciculation of the muscles and zoster eruption are not symptoms but signs. The author is thrifty with the use of eponyms, but he could cut the quota still further. Why the syndrome of Weber, Millard-Gubler, and Foville? Why the reflex of Hoffmann, Bechterew-Mendel, and Oppenheim? Why the sign of Wartenberg? A physiological or an anatomic name will mean more to the student than any proper name, however illustrious. The term "Parkinsonism" has already led to such a horror as "Rombergism." It is better to drop the "ism" completely; these are diseases or signs, not doctrines, tenets, or tendencies. There are no "tendon reflexes," but deep muscle reflexes, and they should be named according to the muscle involved. They should be called reflexes consistently and not "reflexes" in one place and "jerks" in another. There is no supinator jerk or radial reflex," but there is a brachioradial reflex. Both "paresthesiae" and "dysesthesiae," are referred to, but spontaneous sensations cannot be called paresthesiae, since "para" means beside or added to. Paresthesiae therefore imply an external stimulus. There cannot be a "nervus cutaneous antibrachii lateralis." It is ante, meaning "before" and not "anti," which means against. By passive movement we examine the muscle "tonus," not the muscle "tone." There is an acoustic muscle tone, besides the muscle tonus. Though the term "superficial abdominal reflex" is used once, later the confusing term "abdominal reflexes" is used without further elaboration and without distinction between superficial and deep abdominal muscle reflexes. The term "pseudobulbar palsy" is called "unsatisfactory," but it is still used. There is nothing "pseudo" about this palsy; it is genuine. It should be called "spastic" or "supranuclear." Spastic spinal palsy is not called "pseudospinal palsy." Why then should a spastic bulbar palsy be called "pseudobulbar palsy?" One gets the impression that meralgia paresthetica is a traumatic affection; however, it is more often nontraumatic than traumatic. Speaking of painful feet, it is said: "This syndrome has been attributed to deficiency of nicotinic acid, or of pantothenic acid." The comma should not be there, since the two kinds of acid are different.

Frontal Lobotomy and Affective Behavior: A Neurophysiological Analysis. By John F. Fulton, M.D., Sterling Professor of Physiology, Yale University, New Haven. [Thomas William Salmon memorial lecture.] Cloth. $3. Pp. 159, with 21 illustrations. W. W. Norton & Company, Inc., 101 Fifth Ave., New York 3, 1951.

The author is eminently equipped to discuss frontal lobotomy from the physiological viewpoint. As the result of animal experimentation in the laboratory under the author's direction, Egas Moniz, in 1935, conceived the idea that leukotomy might be of value in the treatment of mental disease. Moniz developed his clue rapidly into a form of treatment which has now been used on at least 20,000 persons in many different countries. The historical background of this method of treatment and the physiological function of the cerebral cortex discussed by the author in the first part of the book form the subject of the first two Salmon Memorial Lectures given at the New York Academy of Medicine, now available in printed form. A subsequent chapter deals with recent behavioral studies in animals experimentally operated on, and the terminal section of the book considers the operation in man. The author believes that frontal lobotomy is an efficacious therapeutic tool for the relief of symptoms in patients with otherwise hopeless psychoses. In the past three years, to overcome the difficulties that arose when the operation was used without full recognition of the basic physiological principles behind it, surgeons have adopted a restricted procedure and have attempted to fit the operational technique to the nature of the mental illness which they hope to overcome. Depressed patients and schizophrenic patients with subnormal psychomotor activity should, in the author's opinion, have a resection of the superior orbital gyrus or an interruption of its projections; the agitative, aggressive, and overactive psychotic patient should be treated by singulectomy or topectomy involving areas 9 and 10 of the cerebral cortex. Operations involving the orbital surface of the brain do not result in intellectual impairment. The modern tendency is to replace radical lobotomy, as originally carried out in 1935, by the method of a more circumscribed lesion.

This book is sound from many points of view. The physiological approach is essential to a clear understanding of this relatively new surgical procedure. The conservatism expressed by the author is admirable and should be given great weight in decisions regarding the use of lobotomy in the treatment of mental disease. This book is clearly written and contains adequate illustrations and a selective bibliography. It gives a succinct summary of the problem; however, for the final clinical evaluation of frontal lobotomy, we must wait until some future date.

Tumors of the Skin: Benign and Malignant. By Joseph Jordan Eller, B.S., M.D., Director of Department of Dermatology, New York City Hospital, New York, and William Douglas Eller, M.D., Assistant in Dermatology and Syphilology, University Hospital, New York-Bellevue Medical Center, New York. Second edition. Cloth. $15. Pp. 697, with 553 illustrations. Lea & Febiger, 600 S. Washington Sq., Philadelphia 6, 1951.

In this volume, the authors have classified and described, clinically and histologically, more than 100 types of tumors of the skin and adjacent mucous membranes. Discussions on etiology, differential diagnosis, and various modes of therapy are included. The first half of the book is devoted to benign tumors and precancerous lesions of the skin; the last half discusses malignant tumors and treatment. For the commoner tumors, complete details of therapy are given, including techniques of scalpel or electrosurgery, dosage schedules for roentgen ray and radium therapy, and any indicated medical or physical treatment. In the chapter on carcinoma of the skin, there are helpful diagrams illustrating placement of radium and the optimal zone of roentgen irradiation or excision in typical lesions of different sites. A special chapter deals with techniques of plastic surgery as they apply to excision of skin tumors. General discussions of radi-

ation therapy and radiation physics are included in the section on carcinoma of the skin and in a separate chapter at the end of the book. A number of dosage tables and diagrams are included. An extensive bibliography follows each chapter, and the material is well indexed. In this edition, several new disease entities have been added, and the sections on therapy have been expanded and brought up-to-date. This is a useful reference book for dermatologists, surgeons, roentgenologists, and others interested in tumors of the skin, and it should be particularly helpful as a manual of treatment for the dermatologist and general practitioner.

Surgery of Peripheral Nerves. By Emil Seletz, M.D., F.A.C.S., F.I.C.S., Assistant Clinical Professor of Neurological Surgery, University of Southern California School of Medicine, Los Angeles. Art editor: Tom Jones, Professor of Medical and Dental Illustration and Head of Department, University of Illinois, Chicago. Cloth. $10.75. Pp. 185, with 136 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto, 2B, 1951.

This volume is primarily an illustrative atlas covering the surgical technique involved in suturing disrupted peripheral nerves in every possible location. The author, during World War II, had experience with over 2,000 nerve injury patients at Wakeman Hospital Center, and he is well qualified to delineate the results of this experience. Skin incisions that permit the exposure of each major nerve in all locations are illustrated. When incisions in relation to moving joint surfaces are unavoidable, they are placed so as to produce a minimum of subsequent disability from scar contracture. Techniques are illustrated for mobilizing long segments of nerves to overcome the large gaps between the nerve ends that often occur in war wounds. A continuous incision over the length of the entire nerve trunk is not recommended; skin and subcutaneous scarring are reduced by interrupted incisions. Mobilization of normal nerve is then accomplished by undermining intact superficial tissues and fascia. These refinements in technique developed independently in the hands of many army neurosurgeons, but Dr. Seletz deserves credit for being the first to emphasize them in the literature. The actual operative photographs are not technically good because of poor contrast. The schematic illustrations of surgical anatomy are pleasing but add little to those available in standard texts.

The diagnosis of peripheral nerve involvement is covered by photographs of standard deformities, with outlines of typical sensory loss, and the muscles that might be affected are listed with the expected deficit in movement. Numerous anatomic drawings do not greatly assist in illustrating the practical motor deficits to be brought out by clinical testing. No attempt is made to delineate those functional tests that are useful in practice from those that give equivocal results because of substitution by uninvolved muscles. Therefore, as a reference for diagnosis of nerve injuries, it is believed that this volume compares unfavorably with the excellent manuals already available.

The technique for suture of the grossly disrupted nerve is clearly described; however, a neuroma in continuity is the more frequent finding in military wounds, and advice to the surgeon regarding choice of treatment in this situation is lacking. Exposure of lesions high in the brachial plexus or sciatic foramen is demonstrated without mention of the minimal benefit that is derived from nerve suture in these situations. Despite these criticisms, this volume will constitute a useful reference for those undertaking the surgical repair of peripheral nerve lesions.

Cardiac Pain. By Seymour H. Rinzler, M.D., F.A.C.P., Instructor in Rehabilitation, New York University College of Medicine, New York. Publication number 113, American Lecture Series, monograph in American Lectures in Circulation. Edited by Irvine H. Page, M.D., and A. C. Corcoran, M.D. Cloth. $3.75. Pp. 139, with illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto, 2B, 1951.

This brief lecture is concerned with the history of knowledge of cardiac pain, the nerve pathways of the heart, the pathogenesis and differential diagnosis of cardiac pain, and some common medical and surgical techniques for the relief of this condition. There are already many excellent, scholarly volumes that deal with this subject.

Angiocardiography. By Charles T. Dotter, M.D., Assistant Professor of Radiology, Cornell University Medical College, New York, and Israel Steinberg, M.D., Assistant Clinical Professor of Radiology and Medicine, Cornell University Medical College. Annals of Roentgenology Series of Monographic Atlases, Volume XX. Cloth. $16. Pp. 304, with 635 illustrations. Paul H. Hoeber, Inc. (medical book department of Harper & Brothers), 49 E. 33rd St., New York 16, 1951.

This book is an authoritative reference based on several thousand angiocardiographic examinations by the authors, whose experience with the method doubtlessly exceeds that of any other group in the field. It deals comprehensively with the technicque, indications, limitations, and interpretations of angiocardiography in the normal heart as well as all types of congenital and acquired heart disease. In addition, examination of pulmonary disease and mediastinal tumors by contrast visualization is also covered.

This book will find its greatest usefulness for those who do a considerable amount of cardiac fluoroscopy. In this respect, it will enable the fluoroscopist to give meaning to the cardiac silhouette in terms of its "living anatomy," rather than to employ the empiricism of inflow and outflow tract enlargement. Although angiocardiography in congenital heart disease is dealt with comprehensively, it seems likely that the technique will find less and less diagnostic use as cardiac catheterization is more extensively employed.

The book is profusely illustrated with reproductions of angiocardiograms, accompanied by explanatory line diagrams that are invaluable. The style of the text is concise and readable but, at times, unnecessarily repetitious. The authors are to be complimented on presenting a specialized subject in a lucid and appealing manner.

The People in Your Life: Psychiatry and Personal Relations. Edited by Margaret M. Hughes. By ten leading authorities. Prepared under auspices of Town Hall, New York. With foreword by Isabel Leighton. Cloth. $3.50. Pp. 278. Alfred A. Knopf, Inc., 501 Madison Ave., New York 22, 1951.

This is a compilation of a series of lectures on human relationships. The lectures were prepared and delivered by leading authorities in such fields as psychiatry, sociology, religion, and anthropology, and were discussed on the "Town Hall of the Air" radio program. The purpose of the program was to advance mental health by discussion of some of the most important and often troubling situations in an average human life.

Chapters of the book are complete presentations in themselves, including a "question and answer" period of audience participation. They are arranged to cover the cycle of living, from the first interrelationships of men and women through problems of establishment of families, growth of children, and correlated problems of business contacts, personal friendships, and social contacts.

The material discussed is pointed and readable, and the questions and discussions are valuable. This book will be of interest to both medical and lay persons. It is one of the best collections of material on preventive aspects of mental health for general consumption that has appeared in recent years.

Studies in Medicine: A Volume of Papers in Honor of Robert Wood Keeton. Cloth. $8.50. Pp. 396, with illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto, 2B, 1951.

This volume of interesting and stimulating papers was prepared in honor of Dr. Robert Wood Keeton and was presented to him on the occasion of his retirement as head of the department of medicine of the University of Illinois College of Medicine, Aug. 31, 1951. The book was made possible by contributions from many of his friends, associates, and students, and the wide range of topics covered reflects his diversified interests.

The profession of medicine reaches its height in men who not only have contributed knowledge that saves and prolongs lives and mitigates suffering but also possess the graces and refinements of life that constitute exemplary character. It is such men who, through the ages, have provided support for the claim that the mission of the physician is of the noblest kind. The many articles in this book are a token of the fact that Dr. Keeton became a part of all whom he met. Prepared by colleagues to celebrate his distinguished career in academic medicine, it is a

reminder that the qualities of quiet dignity, unfailing courtesy, gentleness, and consideration for others have earned for him the respect and admiration of students, faculty, and professional associates alike.

Rosenau Preventive Medicine and Hygiene. By Kenneth F. Maxcy, M.D., Dr.P.H., Professor of Epidemiology, Johns Hopkins University, School of Hygiene and Public Health, Baltimore. Seventh edition. Cloth. $14. Pp. 1462, with illustrations. Appleton-Century-Crofts, Inc., 35 W. 32nd St., New York 1, 1951.

The seventh edition of this well-known book is a great improvement over the sixth edition, which appeared in 1935 and was in serious need of revision. The principal revising author is Dr. Kenneth F. Maxcy, school of hygiene and public health, Johns Hopkins University, but he is helped in this work by about 25 collaborators. The book is intended as a reference book rather than a textbook, except, possibly, in the case of advanced students. The principal author and collaborators are capable persons, and there seems to be no instance in which there is reason to doubt the facts or the conclusions that have been reached.

Many of the broader aspects of the Rosenau book have been lost, for example, the excellent discussion on the subject of genetics in previous editions. A book of this size can hardly be reviewed completely in a short space, but it seems adequately to cover the subject indicated by the title. It should be at the elbow of every person who does public health work, but it is rather doubtful if it will become the "Bible" of public health, as were the other Rosenau volumes in their time. It has both the advantages and the disadvantages that are to be found in any book that is essentially a compilation of learned articles on various technical subjects, written by persons who are specialists in the particular field covered.

The Rise of Scientific Philosophy. By Hans Reichenbach. Cloth. $3.75. Pp. 333. University of California Press, Berkeley 4; Cambridge University Press, Bentley House, 200 Euston Rd., London, N.W.1, 1951.

In spite of the disdain with which some medical men look upon current philosophy, this volume would be helpful to the intelligent physician who is interested in a brief consideration of the significant developments of recent philosophy. As the author says, "This book is written with the intention of showing that philosophy has proceeded from speculation to science." Following the criticism of the speculative philosophy of the past, Professor Reichenbach shows how modern philosophy is being built on the firm foundation of agreements reached as a result of scientific study. Significantly, it is implied that scientific knowledge of ourselves and our environment places limits to what we may believe. We are shown how to find our way through our world without resorting to unjustifiable beliefs or assuming supernatural origin for moral standards. Modern logic is considered in detail, with an analysis of the nature of ethics. Indication is given of the practical help of symbolic logic in certain types of brain injury. The discussion on ethics is particularly helpful to the alert physician. Unfortunately, the volume is not documented, so that there is little incentive for the reader to browse further. Nevertheless, the book is an excellent presentation of the potential development of modern scientific philosophy.

After the A Bomb: Emergency Care in Atomic Warfare. Edited by Charles F. Behrens, M.D. Cloth. $2.50. Pp. 182, with 5 illustrations. Thos. Nelson & Sons, 19 E. 47th St., New York 17; Parkside Works, Dalkeith Road, Edinburgh 9; 3 Henrietta St., London, W.C.2, 1951.

According to its editor, this book is intended to provide a concise review of medical responsibilities at the working levels applicable to disaster conditions. Generally, it can be said that the book accomplishes this purpose.

With the vast amount of material that has been written within the last few years regarding the medical aspects of atomic, biologic, and chemical warfare, it is not surprising to find relatively little that is new in a book on these subjects. While it might be argued that the seriousness of the items under discussion warrants repetition, it must be recognized that the effectiveness of the book is reduced because of it.

By obtaining contributions from various authorities on the different subjects discussed, the editor has insured the technical accuracy of his work; however, this system has resulted in a certain amount of repetition, a lack of continuity, and a shifting of emphasis. In addition, whereas some of the chapters are extremely general, others are equally specific. It would appear that the material covered in chapter four, dealing with "Care of Burns and Other Trauma as Related to Mass Casualties," warrants considerably more space and emphasis.

Unfortunately, the basic premise on which all of the articles are predicated is undeniably true; that is, that the medical profession as a body is inexperienced in the care of atomic injuries and is unprepared to cope with an atomic explosion with its resulting hundreds of thousands of human casualties. Therefore, if the book serves to further acquaint the profession with the medical implications of an atomic attack and awakens in the individual physician an awareness of his responsibilities in this atomic age, it will have served a decidedly useful purpose.

Disorders of the Heart and Circulation. Edited by Robert L. Levy, M.D., Professor of Clinical Medicine, College of Physicians and Surgeons, Columbia University, New York. Cloth. $12. Pp. 944, with illustrations. Thos. Nelson & Sons, 19 E. 47th St., New York 17 [medical book publishing business purchased by Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2], 1949-1951.

This is a compilation of 34 articles by 41 authors, all well known for work in their respective fields. The chapters, which have also appeared in "Nelson's Loose-Leaf Medicine," have been combined to make this book. The field covered is cardiology, and about three-fourths of the contents are concerned with the heart and its diseases; however, there are also six chapters dealing with arterial and other diffuse diseases. There is no chapter on the veins or the capillaries, a reflection not on the book but on the narrowness of current medical views on the circulation and its abnormalities. The chapter on clinical electrocardiography, almost one hundred pages long, is a monograph in itself; it discusses the value of the electrocardiogram and emphasizes its limitations.

Most of the authors belong to the descriptive rather than the experimental school, and this is clearly reflected in their articles. There is a commendable interest in the medical history of the various fields. This book is up-to-date, and the new data on cardiac output and pulmonary pressures in health and disease are discussed at length, as are the newer surgical techniques for the correction or alleviation of cardiac abnormalities and hypertension. The book is well written, and the illustrations are excellent. The bibliographies are variable; some, like that following Maud Abbott's article, are extensive, while others might be criticized as meager. This book is strictly a reference book, almost too heavy to hold comfortably in the reading position and far too detailed for students; however, it is well worth a place in the library, and physicians interested in diseases of the heart and circulation will probably consult it frequently.

Selective Toxicity with Special Reference to Chemotherapy. By Adrien Albert, D.Sc., Ph.D. Medicine, F.R.I.C., Professor of Medical Chemistry in Australian National University, Melbourne. Cloth. $1.75. Pp. 228, with 36 illustrations. John Wiley & Sons, Inc., 440 Fourth Ave., New York 16; Methuen & Co., Ltd., 36 Essex St., Strand, London, W.C.2, 1951.

This subject has grown in scientific and commercial importance during the present century. The advent of certain weed killers and insecticides is an outgrowth of the use of selectively toxic agents, which injure undesirable forms of life without too seriously damaging the desirable forms. The author has chosen to review the toxicants, not in their order of economic importance but rather as to how well understood their action might be. This is defended on the ground that the mechanisms by which selectively toxic agents may act are limited; therefore, study of the known mechanisms of action may enable development of new approaches.

The author's style is clear and concise, and the material is discussed in an interesting and thought provoking sequence. The importance of comparative biochemistry as the key to revelation of metabolic differences that lead to the discovery of selectively toxic materials is developed soundly. The physicochemical and pharmacologic approaches to chemotherapy are also discussed in some detail. This is an interesting little book for the scientist desiring information on the background of selective toxicity.

QUERIES AND MINOR NOTES

INFLUENZA

TO THE EDITOR:—*Is there anything new for the treatment of influenza? Many physicians are using antibiotics, without much improvement. In a few cases in which antibiotics were used, the patients passed bloody urine and contacted dermatitis, especially after taking penicillin.* *M.D., Arkansas.*

Note: This inquiry has been referred to two consultants, whose respective replies follow.—ED.

ANSWER.—Thus far there is no remedy that has received general approval. There have been favorable reports in regard to the use of aureomycin in the treatment of influenza. Others have expressed doubt concerning its value for that purpose. If aureomycin is not helpful, it may be well to try terramycin.

ANSWER.—Influenza, as it has been observed in recent years, occurs in explosive outbreaks, often simultaneously in widely scattered areas. The patient is usually ill from three to seven days and has symptoms of headache, fever, chills, malaise, cough, generalized aching, and weakness. The fatality rate has been low in late years and almost entirely confined to the older members of the population. The cause has been accepted as a filtrable virus of several types, A, A′, and B. In 1950 and 1951, the A′ type was most frequently identified. Since the clinical picture can resemble that of a number of other respiratory infections, a definite diagnosis can be assured only by isolation of the virus from the patient or by the significant rise in antibody titer in serum samples taken early and late in the course of the disease. There is no known therapeutic agent or antibiotic that is effective in the disease caused by any of the types of influenza virus; however, since the greatest part of the mortality results from bacterial complications in the respiratory tract, patients with a severe influenza-like illness should receive appropriate antibiotics.

TREATMENT OF CALCINOSIS OF KIDNEYS

TO THE EDITOR:—*A man, 26 years of age, has a kidney condition, which dates back 15 years. The diagnosis is calcinosis of the kidneys. The blood creatinine is 9.4 mg. per 100 cc., and the urea nitrogen is 46 mg. per 100 cc. The specific gravity of the urine is 1,008. Albumin is 1+ or more. What treatment might help? What might he be given to increase the excretion of urinary solids? Would transplantation of kidneys be advisable?* *Thomas Moe, M.D., Moose Lake, Minn.*

ANSWER.—If a patient 26 years old has neither anemia nor hypertension with chronic renal disease, the prognosis would be fairly good in spite of elevated creatinine and urea levels in the blood. There is no known way to increase renal blood flow and filtration in such a patient, and the implantation of a human kidney, which never functions longer than two months, or use of an artificial kidney should be postponed until blood urea nitrogen is over 100 mg. per 100 cc., and the patient is clinically in uremia. Meantime, on a low protein diet with adequate salt and calories, well-being should be maintained for a long time.

IRON AND BLACK STOOLS

TO THE EDITOR:—*Is it possible to administer iron orally in a dosage that will be effective in the treatment of secondary anemia and yet will not discolor the stools?*

Daniel Haffron, M.D., Elgin, Ill.

ANSWER.—It is practically impossible to administer therapeutically effective doses of iron for secondary anemia without discoloring the stools.

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

COLD DISINFECTION OF INSTRUMENTS

TO THE EDITOR:—*Is 5.25% sodium hypochlorite solution ("chlorox") of any value for cold sterilization of instruments in an office? What strength formaldehyde solution is advised?*

M.D., Ohio.

ANSWER.—Cold disinfection of instruments is not recommended unless heat sources (electricity and gas) are not available. If chemical disinfectants are employed, instruments should previously be freed from organic matter. If the instruments are to be used on sensitive tissues, residual amounts of toxic chemicals must be removed.

Commercial sodium hypochlorite solutions contain 2 to 6% available chlorine and are supplied with varying degrees of alkalinity. The antibacterial activity of sodium hypochlorite diminishes with increased alkalinity, because it is dependent on the formation of hypochlorous acid and available chlorine. Solutions of sodium hypochlorite are instable, corrosive, and toxic; "stabilized" hypochlorites are less effective antibacterials. While the hypochlorites and other chlorine compounds are widely recognized as valuable agents for sanitizing processes, they are not recommended for the disinfection of the physician's instruments.

A number of satisfactory formulas of noncorrosive formaldehyde solutions have been proposed for chemical disinfection, including the following: 69% isopropyl alcohol, 1% methyl alcohol, 0.1% sodium nitrite, 0.8% sodium bicarbonate, and water in a quantity sufficient to make 100%. Formaldehyde solutions are subject to many of the limitations of other chemical disinfectants.

TRANSMISSION OF DEAF MUTISM

TO THE EDITOR:—*A young man married a girl whose mother has a deaf mute sister. What are the chances of the condition being transmitted to his offspring?* *M.D., Illinois.*

ANSWER.—Deaf mutism is attributed to severe deafness before speech is acquired, so that the person does not learn to speak without specialized training. In fewer than half the cases the severe deafness is endogenous, due to an inherited defect in the cochlea of the inner ear. In such cases there are often other persons in the family with a similar condition. In more than half the cases the profound hearing impairment is exogenous: that is, it is due to a toxic or infectious agent in utero or in early infancy, which destroys or severely damages the hearing nerve. For example, rubella in the mother during the first three months of pregnancy not infrequently causes deafness in the child. If there are other persons in the family who were deaf mutes, the tendency could be transmitted from a normal hearing parent to her child. This possibility would be greatly enhanced if the mother and husband were cousins. If there are no other cases in the family, it is quite possible that the wife's sister suffers from an exogenous deafness, which would not be transmittable.

REMOVAL OF ACNE SCARS

TO THE EDITOR:—*I would like information on the technique of carbon dioxide snow-ether application for the removal of acne scars from the face.*

Leo R. Hawkes, M.D., Preston, Idaho.

ANSWER.—This treatment has few advocates, for the results are not satisfactory. One method of preparing the mixture is as follows: A piece of dry ice is pulverized with a mortar and pestle. Precipitated sulfur is added, and finally acetone is added in small amounts as the mixture is stirred into a smooth slush. The solution is applied to the skin with an applicator made of a ball of cotton firmly encased in gauze. The length of application and amount of pressure necessary must be determined by experience. Strong and prolonged pressure, besides causing pain, will produce a blister.

PROSTATIC CANCER

To the Editor:—*Acute urinary retention suddenly developed in an ambulance driver, aged 65. He was hospitalized, and a suprapubic prostatectomy was performed. The pathological diagnosis was adenocarcinoma of the prostate gland. Several weeks later, bilateral orchiectomy was performed. After an asymptomatic interval of four years, severe sacral and sciatic pain and weakness of the lower extremities developed, especially while climbing stairs. At present, six years after prostatectomy and orchiectomy, he exhibits bilateral foot drop, 2+ pitting edema of the pretibial area, and absent patellar and ankle reflexes. A roentgenogram of the pelvis reveals discrete nodular lesions of the lower lumbar vertebrae and pelvic bones, with loss of trabeculae in involved areas. The following laboratory results were obtained. Serum phosphorus was 8.2 mg. per 100 cc.; acid phosphatase was 20.1 units, and alkaline phosphatase was 15.5 units. The urinalysis showed normal results. The hemoglobin was 60%; total proteins were 7.6 mg. per 100 cc.; albumin, 5.1 mg. per 100 cc.; and globulin, 1.8 mg. per 100 cc. The patient is bedridden and can move about only with assistance. 1. What results can be anticipated from the use of estrogens? 2. Can the level of phosphatase activity be used as a reliable index in judging therapeutic response? 3. Is roentgen ray therapy indicated? 4. What is the present status of nitrogen mustards and urethane as palliative treatment in these cases? 5. Is edema probably caused by obstruction of venous return by pelvic metastatic lesions?*

Cromwell C. Douglas, M.D., Norfolk, Va.

This inquiry was referred to two consultants, whose respective replies follow.—Ed.

Answer.—1. Estrogen therapy may still be effective after orchiectomy and may produce a remission of tumor growth in this patient. An adequate trial would certainly be indicated. 2. The serum acid phosphatase level will serve as a fairly accurate index of the activity of the lesions outside the prostatic capsule. A fall to normal limits should be accompanied by clinical improvement, which, in itself, is probably the best index of response. A persistently high acid phosphatase level would indicate a poor prognosis. 3. Roentgen ray therapy directed to the bony lesions in the spine and pelvis is often helpful in the relief of pain, and its use might be of temporary benefit to this patient. 4. The use of nitrogen mustard and urethane has been abandoned by most clinics because of toxicity and poor response in the majority of patients. 5. Yes.

Answer.—The situation described seems to be of a terminal nature. The use of estrogens is definitely indicated, but, in view of the length of time since bilateral orchiectomy, it is questionable as to whether any response can be anticipated. The level of phosphatase activity will probably not be altered by the administration of estrogens. Roentgen ray therapy has proved to be of considerable value in many of these cases. Frequently, it reduces the pain, making the patient more comfortable. In a few instances, there has been some shrinkage of the metastatic tissue itself, but the greatest value lies in the relief of pain. As yet, there has been no definite report on the value of nitrogen mustards. Urethane has been used with variable results; its actual value is questionable. The foot drop, the pitting, the edema, and the various neurological manifestations are probably due to the metastatic lesions.

IRRADIATION FROM TELEVISION RECEIVERS

To the Editor:—*Please discuss the irradiation hazard to men repairing television receivers.*

James E. Cameron, M.D., Alexander City, Ala.

Answer.—Large television units, such as those used in motion picture theaters, operate at a kilovoltage as high as 100. A large number of roentgen rays are produced. Such tubes are housed in a lead lined cabinet. There would be a definite hazard if repair men were exposed to the unguarded tube while it was in operation. In domestic television units there is no radiation hazard if the tubes are operating at normal voltages. If the voltages exceed the normal by 100 to 200%, long wave roentgen rays would be emitted, which constitute a hazard for a short distance from the tube.

HYPERTROPHY OF BOTH BREASTS

To the Editor:—*A girl, aged 19, has marked "virginal type" hypertrophy of both breasts. She would like to have surgery done to remedy the condition. Please tell me whether subsequent pregnancies will have any adverse effect on her and/or the breasts, allowing for successful surgery and good cosmetic results.*

Henry Rosner, M.D., Brooklyn.

Answer.—Mammary hypertrophy originates shortly after the onset of puberty, and progressive growth may last several months or several years. Growth usually ceases, but there may be a new spurt during pregnancy. Although some abnormal hormonal stimulus has been suspected, the cause of excessive growth is not known. If breast enlargement is so marked that it impedes normal activity, in spite of good support, or creates an emotional problem in the young woman, surgery may be indicated. Although for cosmetic reasons some physicians prefer to retain parts of the glands, most favor complete removal lest the remaining tissue subsequently become reactivated, especially during pregnancy. If some breast tissue is retained, pregnancy need not offer a serious problem, for engorgement can be suppressed by hormones.

NASOPHARYNGEAL FIBROMA

To the Editor:—*I would like information regarding endocrine therapy for a 15-year-old boy who had a juvenile nasopharyngeal fibroma removed four weeks ago.*

M.D., California.

Answer.—The idea has developed, in some quarters, that nasopharyngeal fibromas tend to resolve at puberty and that estrogen therapy may be valuable; actually, however, these tumors usually do not disappear spontaneously at puberty, and estrogen therapy is of little value. The tumors occur primarily in the male and do not respond to irradiation. Operative removal is generally considered to be the treatment of choice, although, in some instances, the implantation of radium is effective. These tumors are not malignant, but they tend to recur. They are important clinically, because they are space filling lesions, and hemorrhages from them are common; in fact, hemorrhage is one of the common complications of operative removal. Enough bleeding may occur without treatment to cause a definite reduction in the concentration of hemoglobin in the blood.

UNDESCENDED TESTIS

To the Editor:—*1. Is it possible for a testis to be absent? At an operation for unilateral undescended testis, search for the testis in the retroperitoneal and retropubic area failed to reveal any evidence of it. 2. Is it possible that the testis was so rudimentary that it could not be distinguished? There were vessels found in the inguinal canal leading down into the scrotum and upward to the retroperitoneal area attached to the peeled back peritoneum. 3. Do the internal spermatic vessels accompany the descent of the testis, or do the vessels and the testis develop and descend independent of each other?*

M.D., New York.

Answer.—1. Yes. The testis may be absent, although it should be borne in mind that an undescended testis may be found anywhere along the posterior abdominal wall up to the level of the kidney. 2. It is possible that the testis is not recognized, although, it usually is. 3. The internal spermatic vessels accompany the testis in its descent.

EOSINOPHILIC GRANULOMA OF BONE

To the Editor:—*Please discuss the use of corticotropin (ACTH) or cortisone in the treatment of eosinophilic granuloma of bone.*

M.D., Pennsylvania.

Answer.—Corticotropin (ACTH) and cortisone have been used in the treatment of localized eosinophilic granuloma of bone without notable results. These drugs have not produced remissions similar to those accomplished in so treating eosinophilic infiltration of the lungs (Loeffler's syndrome). Local excision, if feasible, and/or roentgen ray therapy continue to be the treatment of choice for localized eosinophilic granuloma of bone.

SUBSTERNAL THYROID ADENOMA

TO THE EDITOR:—*What are the symptoms of substernal thyroid adenoma? Is it possible for adenoma of this type to cause symptoms without being large enough to be seen on a chest roentgenogram?* *B. A. Lucking, M.D., Helena, Mont.*

ANSWER.—Most substernal or intrathoracic goiters cause no symptoms unless they are toxic or unless they enlarge to such a degree that they cause compression of the trachea and great vessels at the level of the thoracic inlet. Pressure from the expanding goiter may manifest itself by causing dyspnea, distention of the cervical veins, with development of collateral circulation visible over the upper portion of the chest, and a peculiar cavernous sound when the patient breathes deeply with his mouth open. The roentgenogram is accurate in diagnosing the presence of thyroid adenomas that have entered the thoracic cavity. Lateral views and anteroposterior views should be obtained. It is fair to say that it is impossible for a thyroid adenoma to be large enough to cause pressure symptoms without being clearly visible on the roentgenogram. Displacement or compression of the trachea is regularly observed in large intrathoracic goiters; dysphagia is a rare symptom. Choking sensations are usually the result of globus hystericus and are not due to organic disease of the thyroid.

MORPHINE INJECTION USING UNSTERILE TABLET

TO THE EDITOR:—*There is a widespread practice of placing an unsterile tablet of morphine or codeine in a sterile syringe, drawing up sterile water, and injecting without boiling after dissolving. I have never heard of an infection resulting from this practice. Could this be due to possible germicidal action of the morphine or codeine?*

Robert S. Srigley, M.D., Hollis, Okla.

ANSWER.—The concentration of morphine sulfate (15 mg.) dissolved in 1 ml. of water is not sufficient to have a germicidal effect by salt action. A 60 mg. dose of codeine sulfate in 2 ml. might have a germicidal effect. Large doses of heroin and milk sugar do not have a germicidal effect as is shown by the occasional transmission between addicts of malaria, staphylococcal infections, and other diseases.

The routine practice of placing an unsterile tablet in sterile water for injection probably does not produce local infection because of two factors. 1. The tablets are made by adding enough alcohol to lactose and opiate to form a workable triturate. The tablets are then pressed out and packaged without contamination. 2. Morphine and codeine have a strong whealing (persistent histamine-like) effect, which may increase the capillary circulation at the injection site. This would decrease the localization of injected pathogens.

THE OPHTHALMOSCOPE

TO THE EDITOR:—*How much enlargement is obtained when using the +70 lens of an ophthalmoscope?*

Walter Weinberger, M.D., Juniata, Altoona, Pa.

ANSWER.—It is difficult to conceive of an eye with 70 D. of hyperopia. In most ophthalmoscopes, a +20 lens is the most convex, and 50 D. is the most that can be obtained with the Friedenwald ophthalmoscope; however, regardless of the power of the lens in the ophthalmoscope, the magnification will be about the same, if the lens corresponds to the amount of ametropia and if the observer is at the anterior focal plane of the subject's eye. When the emmetropic observer views the fundus of an emmetropic subject, parallel rays of light fall on the observer's retina, and the image formed is the same size as the object. The image on the observer's retina is projected into space to the point of clear vision (20-25 cm.). This distance varies with the observer and thus the magnification also varies. If this point is 22 cm. and the distance from the nodal point of the eye to the retina is 15 mm., then, by similar triangles, the magnification is found to be about 15×. In practice, the ophthalmoscope is usually in front of the anterior focal plane of the subject's eye. A lens weaker than the actual amount of hyperopia is used, so that the magnification is slightly less than that in emmetropia.

CRACKS AT CORNER OF MOUTH

TO THE EDITOR:—*In regard to the query concerning the relationship of dentures and cracks at the corner of the mouth,* (J. A. M. A. **147:**1508 [Dec. 8] 1951) *I believe that amplification of the answer given would be helpful in obtaining a cure of the patient's affliction. Although well-fitting dentures will not produce cracks at the corners of the mouth, removal of these dentures on retiring at night frequently results in the accumulation of an excessive amount of saliva at the angles of the mouth. This moist environment frequently results in a combined streptococcic and monilial infection. The retention of the dentures overnight and the use of an antiseptic ointment, such as 5% ammoniated mercury ointment, after each meal and at bedtime might effect a cure in this patient who had insufficient benefit with vitamin therapy.*

Bernard H. Winston, M.D.
324 Professional Building
11th and Grand Ave., Kansas City 6, Mo.

TO THE EDITOR:—*In the reply to the letter concerning cracks at the corners of the mouth, as published in Queries and Minor Notes, Dec. 8, 1951, we feel that Dr. Hackett is dealing with a "pseudocheilosis," resulting from a decrease in the intermaxillary space or vertical dimension caused by the upper denture. Subjective comfort on the part of the patient is not indicative of a well-fitting denture. Because of accommodation the patient may be perfectly happy with a denture, even though the intermaxillary space is greatly decreased.*

The lesions produced by a reduction of the vertical dimension are apparently the result of the upper lip overlapping the lower, producing a fold at the corners of the mouth.

Moisture from retained saliva in this fold, together with bacterial and mechanical irritation, produce the lesions. Most of these lesions disappear when new dentures are made and the correct vertical dimension is established.

Dr. L. W. Burket, in his book "Oral Medicine: Diagnosis, Treatment" (Philadelphia, J. B. Lippincott Company, 1946) noted that the lesions resulting from reduced intermaxillary space slant downward and outward from the corners of the mouth, whereas the true cheilosis, associated with ariboflavinosis, is more horizontal.

Edward C. Dobbs, D.D.S.
Leonard Rapoport, D.D.S.
University of Maryland
School of Dentistry
Baltimore 1, Md.

TO THE EDITOR:—*The question of the relation of dentures to the presence of cracks at corners of the mouth is raised in* THE JOURNAL (**147:**1508 [Dec. 8] 1951). *I should like to point out that in an article entitled "Pseudo Ariboflavinosis" (Ellenberg, M., and Pollack, H.:* J. A. M. A. **119:**790 [July 4] 1942) *a definite relation was shown to exist between the presence of ill-fitting dentures and accompanying perleche and glossodynia; these lip and tongue lesions result from a mechanical defect whereby there is a shortened vertical dimension with overclosure. This leads to secondary fungus infection, usually from Candida albicans. When such a mechanical factor exists, no amount of vitamin therapy will produce any effect. Therapeutic response is achieved when the underlying malocclusion is corrected.*

Max Ellenberg, M.D.
45 East 66th St., New York.

SPHENOPALATINE GANGLION NEURALGIA

TO THE EDITOR:—*In the reply to the query on sphenopalatine ganglion neuralgia in* THE JOURNAL (**148:***161 [Jan. 12] 1952), the consultant makes the following statement: "From 1936 to 1947 not a single reference to this topic appeared in the* Archives of Otolaryngology." *This is not so. In January, 1942, the* Archives of Otolaryngology *published an article by Dr. Watt W. Eagle entitled "Splenopalatine Ganglion Neuralgia."*

Noah D. Fabricant, M.D.,
185 N. Wabash Ave.
Chicago 1.